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No. II.

3. The Distribution of Sleeping Sickness, *Filaria perstans*, etc., in East Equatorial Africa. By CUTHBERT CHRISTY, M.B. and C.M. (Edin.).

4. Adult Forms and Developmental Forms of the Trypanosome found in Sleeping Sickness. By ALDO CASTELLANI, M.D. (Florence).

5. Report on Sleeping Sickness from its Clinical Aspects. By GEORGE C. LOW, M.A., M.B., C.M., and ALDO CASTELLANI, M.D. (Florence). Appendix.—*Filaria perstans* and its Relationship to Sleeping Sickness. By GEORGE C. LOW, M.A., M.B., C.M.

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3.

THE DISTRIBUTION OF SLEEPING SICKNESS, FILARIA PERSTANS, &c., IN EAST EQUATORIAL AFRICA.

By CUTHBERT CHRISTY, M.B. and C.M. (Edin.).

[Preliminary Report, dated Entebbe, Uganda, October 31, 1902.—Received December 5, 1902.*]

(With 3 Maps.)

In studying the Map of the Distribution of Sleeping Sickness, it will be seen at a glance that the disease is connected in some way with the great lake or its waters. In no case has the infection spread far inland, 30 or 40 miles being its limit. It shows no tendency to spread along the Nile source or to other lakes or rivers; 30 or 40 miles, in fact, is beyond the limit. My observations lead me to believe that most cases to be found further than 10 or 15 miles from the lake are cases which have become infected near the shores of the lake. The nearer one approaches the shores of the lake the more prevalent is the disease. Many of the villages by the lake have lost two-thirds of their inhabitants. All the islands in the area are similarly affected. In Buvuma, which I have just recently visited, fully two-thirds of the population have died off, and, at least, half of those now left have the disease; all say, quite cheerfully, that they will get it in time, each day they expect to have the premonitary headache. In this island the mortality has been much aggravated by famine, firstly, the result of drought, and secondly, from dearth of labour, the result of the mortality. The whole island is now almost entirely out of cultivation, except where a few bananas and sweet potatoes are grown by the water's edge. By a cruel irony, one of the most conspicuous

* This more or less informal and preliminary report was, as the date shows, written at an early stage of the labours of the Commission. It has been judged desirable to publish it; but Dr. Christy's full report is printed as No. 6 of this series.

symptoms of this mysterious disease in a large number of the cases is an inordinate appetite till the last, the patient continually waking up and asking for food, when he is so weak that he cannot raise himself upon his elbows. It seems pretty certain that the disease is spreading eastwards. Until I visited Southern Kavirondo it was not known to have spread beyond Usoga, but I found it in its worst form all along the lake shore as far as Kisumu, though less prevalent as I neared that place. Beyond Kisumu, and along the south of Kavirondo Bay, I met with no cases, though a very thickly populated district, till I had passed Homa Bay, and reached Kasagunga and Lusinga Island. Here I again met with the disease, some 10 per cent. or more of the population being infected. The configuration of the map of this district would suggest that the disease, after reaching Uyoma promontory in South Kavirondo, has jumped the mouth of Kavirondo Bay and gone on spreading southwards along the east shores of the lake. From information I gathered, it would seem that there is seldom any communication between the people of South Kavirondo and those on the opposite side of the bay. I learnt that, once in a while, Basoga come in canoes along Southern Kavirondo, cross over to Lusinga Island, or to Kissengere, on the mainland, remain there a day or two only, for the purpose of buying salt, and then return again to Usoga. I hope at a future date to again examine the conditions in these districts.

The history of the disease probably dates back to the beginning of 1900 in Usoga and Eastern Uganda. In Kavirondo, from what I could gather, the disease was not known earlier than 11 months back, and, in Lusinga Island and Kissingere district, 5 months only seemed to be the duration of the epidemic. This, if correct, clearly points to the direction of spread. The duration of the majority of cases is from 5 to 6 months. The infection would seem to be a "place infection," like that of plague. If a house becomes infected, all the inhabitants usually succumb, and, if others go and live in that house, they also become infected, whereas houses in a shamba only a few yards away remain uninfected, although there is communication between the two; but these points have still to be worked out when the distribution problems are solved. The disease is not, and never has been, very prevalent west of Kampala, and no information has yet reached me of its presence on the western shores of the lake. The assertion that it has spread to the Nandi plateau, as far as I am able to ascertain, is incorrect. I have been unable to trace any connection between the occupation of fishing and the disease. Dr. Hodges' fish theory of causation has, however, points in its favour, and I hope in time to make a careful study of the subject. That the disease exists in West Africa we know, and it is possible that it may have been brought to Usoga from the Congo regions—from the Lendu-Congo districts

with the remnants of Emin Pasha's army of Sudanese—but that it has spread gradually across the Continent is not a fact.

If, now, one turns to the Map of the Distribution of *Filaria perstans*, it is seen at once that it in no way agrees, as was supposed, with that of sleeping sickness. As one passes from Usoga into Kavirondo, the percentage of *F. perstans* at once drops from 60 or 70 per cent. to less than 14 per cent., and a little further to 5 per cent. Still further east, beyond the Nzoia River, it is entirely absent, and, in all probability, does not exist elsewhere in the East African Protectorate. Its northern limit, with one exception, is the Usoga boundary, viz., Lakes Choga and Mpologoma, the latter being merely an extensive swamp. The one exception is Mount Elgon. All the slopes of this enormous mole-hill (14,200 feet), except to the north-eastward (uninhabited), are extremely thickly populated. When one's view is not obscured by rainstorms or mist, thousands upon thousands of huts and clearings can be counted dotted about the slopes, which for miles are vivid green, with endless banana plantations, in which the plants grow from 15 to 18 feet high. The Bagesu, the inhabitants of this district on the western and northern slopes of Mount Elgon, are "degraded and simian-like negroes," of small stature and ugly features, which Sir Harry Johnstone says are "perhaps the wildest people to be found anywhere within the limits of the Uganda Protectorate." I did not find them so, for, taking a hint from a neighbouring chief, who sent a message to them explaining who I was, I left my caravan in camp below, and climbed the slopes of the mountain, accompanied only by two interpreters and a porter carrying my blood-testing case. After some patience I was repaid by getting a considerable number of blood slides. I believe few people have ever met the Bagesu of Western Elgon in large numbers on friendly terms, as I did on that occasion. I hope to do so again, for I had no opportunity of examining their dwelling huts, and a knowledge of their domestic life would be of great interest, as possibly throwing some light on the life history of *F. perstans*, for many of the slides taken then contained an enormous number of worms, 100 in one slide being common.

There is no evidence, in my opinion, to point, as has been asserted, to the recent introduction of *F. perstans*, whereas sleeping sickness certainly was recently introduced, or has recurred after the lapse of many years, of which, however, there is no evidence. The first case was reported in April, 1901, from Kampala, though it probably existed in Usoga previous to that date, being masked by the prevailing famine. The suggestion that sleeping sickness will eventually spread over the *perstans* area cannot be put forward.

In passing [northwards] through Usoga the percentage of *F. perstans* remained high (60 to 80 per cent.) up to the last village (Engo), on

the southern shore of Lake Choga. Crossing the lake into Bukedi, the percentage fell to 15 per cent., and two marches further on to 2 per cent. only.

In Bukedi I found from 2 to 4 per cent. of *Filaria diurna* (*nocturna*), and, although the slides containing *F. perstans* were few and far between, all but one of the slides containing *F. diurna* contained also *F. perstans*. Beyond this fact, which, if not a mere coincidence, might point to the probability of the two species being introduced by the same mosquito, I have found no evidence to lead to the belief that the intermediate host of *F. perstans* is a mosquito at all. These few marches through Bukedi, where I touched an area of *Filaria diurna*, which probably extends as far as Egypt, were the only marches during which mosquitoes (a large straw-coloured species, with conspicuously black-banded legs) were a torment, biting all day in thousands, and necessitating a wood fire by the dinner table in the evening.

The distribution map of *F. perstans* is compiled from information gained from blood slides other than those taken on my march through the country.

There used to be great enmity between the Usoga and the surrounding tribes, but there is now free intercourse, so that it is possible that *F. perstans* has only recently extended beyond the borders into Bukedi and Kavirondo by persons who have become infected when visiting Usoga.

On Mount Elgon, although blood slides have been obtained from only one place amongst the Bagesu, I have marked what I think is their area of occupation as equally infected with *F. perstans* as that from which I obtained the slides, as the conditions seemed to be the same. Although I have examined a very large number of blood-slides, I have failed to find *F. perstans* in the blood of sleeping sickness cases living beyond the area marked as that of *F. perstans*; and in the area of both, if a sufficient number of slides are examined, the percentage of *F. perstans* in the sleeping sickness cases always agrees with that of *F. perstans* in the healthy populations.

Southern Kavirondo is an extremely fertile country; the inhabitants are more or less industrious, and enormous quantities of grain are grown, which supply the needs of a vast area to the east and north-east. In passing from this grain country across the border into Usoga the traveller, after crossing the mile strip of no-man's-land, is at once struck by the changed surroundings. In place of the cornfields he finds bright green banana plantations and areas of dense forest with large trees.

Upon studying those boundaries of *F. perstans* as yet mapped out and comparing them with the boundaries of the banana cultivation, it will be seen that the two coincide in a remarkable manner.

This fact must be something more than a coincidence. Wherever the banana is growing, in the districts I have examined, there is found the *F. perstans*. What is more, it would seem that where the banana is most luxuriant (on Mount Elgon) there the inhabitants are most infected (60 to 70 per cent. and the blood crowded). The converse of this is also clearly correct, as will be seen by the map, viz., where the banana is scarce (in Southern Bukedi, and Northern Kavirondo, in which latter place it is only grown inside the mud walls of the small villages) there the percentage of *F. perstans* is small; and where there is no banana there is no *F. perstans*. The banana, I am told, is grown all over Uganda and the Western Provinces (Ankole, Toro, and Unyoro), its limit to the north being the Victoria Nile. It remains for me to prove, if this is so, whether the Nile is here also the limit of *F. perstans*. From slides I have obtained from these districts this would seem to be the case. The banana when fully grown is peeled and boiled or steamed in an earthenware pot. The leaves are used for plates and dishes, for thatching houses, for clothing, for mattresses to sleep upon, for pipe stems and many other things. The skins of the fruit are used for making soap, and the stems for rope, etc.

There is yet another area which coincides in almost as remarkable a manner with that of *F. perstans* as does the area of cultivation of the banana. This is the area in which live people who wear clothes. To the north and east of the red pencil line on the banana map are the Nilotic races to the north of the Victoria Nile, the Bukedi (the word meaning "the land of naked people"), the Bagesu on Mount Elgon (some of whom, however, as I saw them wore small pieces of bark-cloth and skins), and the Kavirondo, who, like the Bukedi, are, both men and women, entirely naked, not even using a loin-cloth. When coming into Kisumu market a small goat-skin, in the case of the men, is occasionally worn over the buttocks, but nothing in front, while some of the women wear a few red and white beads in front and a tassel of string or grass behind. The line of demarcation between the clothed and unclothed is everywhere distinct, but as "civilisation" advances this line will gradually fade with increasing rapidity, as it is now said to be doing. The Victoria Nile therefore has a treble interest attached to it, for it remains to be discovered whether it is the limit of the areas of *F. perstans*, of the banana, and of the clothed people, in a similar manner to that already shown to be the case to the eastward, and mapped out in the attached maps. The word clothing has not, of course, the significance that it has in Europe. It here means merely a loose gown of white Americana, a toga of brown or black native bark cloth (hammered out from the bark of a tree), a specimen of which is enclosed, or a small piece of bark-cloth suspended from the hips. Working in the fields the cloth, in most cases, is discarded.

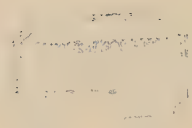
With regard to mosquitoes, I have made a careful collection all

along my route. These are now in the hands of Mr. Theobald of the British Museum (Natural History). There does not seem to be any one species which prevails generally in the *F. perstans* area. The commonest species (? a Stegomyia) was equally common in parts of Kavirondo. The greater part of Kavirondo is high land and open, and the temperature is cool. Mosquitoes therefore at many camps were absent. In no part of Kavirondo, as far as I saw, are mosquitoes a trouble. In Usoga they are more troublesome but nowhere a pest, unless it be as one nears the big swamps surrounding Lake Choga. At most places along the shores of the Victoria Nyanza and in the islands they are seldom noticeable. After crossing Lake Choga into Bukedi they at once become as great a pest as they are on the Niger in West Africa and elsewhere. In the swamps and long grass of Bukedi they are intolerable all day long, and towards evening wood fires are required, the smoke of which is almost as intolerable as the mosquitoes. As one passes eastwards towards Mount Elgon they again become scarce, and on Mount Elgon itself I collected none.

The traveller in Usoga continually has thrust upon his attention pests worse than mosquitoes, viz., bed bugs and body lice. The native of Usoga carries everywhere on his person or clothes a large supply of both, and no amount of ingenuity will prevent the animals from getting from the porter into the load he is carrying. A good supply can always be obtained by searching his piece of bark-cloth, and if more are required hundreds can be found in his hut ensconced in the folds of his papyrus or banana-leaf mattress and elsewhere. In Kavirondo with Kavirondo porters the traveller is free from this annoyance, at least I was, and others I questioned on the subject also had been. The Kavirondo has no clothing in which to harbour the pests, nor has the man of Bukedi. From the few observations I have made, their huts would also seem, for some reason, to be either quite free or less infested than those of the Busoga, but as yet I can make no definite assertion upon this point. The Kavirondo, at all events, make freer use of the universal ammoniacal insecticide—cow-dung—so freely used in West Africa and all over India and other parts of Asia and elsewhere.

DISTRIBUTION OF SLEEPING SICKNESS AS KNOWN UP TO DATE OCT. 23RD 1902. WITH ROUTE MAP.

ALBERT NYANZA
VICTORIA NYANZA
BUGANDA
BUNYORO
BUKEDI
BUSOGA
BUYAGA
BUGANGAZI
BULEMEZI
BUSUJU
BUDDU
KABULA
MAWOGOLA
KIAGWE
KIADONDO
KATEBBE
BUVUMA
KOKI
ANKOLE
BWEKULA
BUTAMBALA
MAWOKOTA
BUSIRO
GOMBA
SINGO
BUKEDI
KIMAMA
BUSOGA
ELGON
KAVIRONDO
NANDI
LUMBWA
KOSOVA
UGAYA
GERMAN EAST AFRICA
GERMAN
Kirota
Masindi
Mubuli
Hoima
Mpala
Bugoma
Bugaya
Kiambalango
Kitangezi
Mukwenda's
Mugema's
Mwenda's
Kiwanga
Kiyanga
Mt Mahorzi
Kakunguru
Kahenda
Kisaasira
Kaheri
Kateta
Bwiro
Kuhagumbus
Naikam
Lake Salisbury
Gedge
Kiboko
Roma
Karuta
Mt Debasien 9500
9700
Kagote
Njera
9300
6750
Suk Mts
Mt Elgon
14200
Gwas Ngishu
6000-7000
Plateau
Mt Sirgoi
Kampi Simba
Kubkong
Kapsabet
Nandi Stn
Nandi Escarpment
Maragolia Hills
Kisumu
Port Florence
Kavirondo Bay
Homa
Lusinga I
Port Victoria
Ukelos
L Gangu
Yala or Lukos
Ugowe
Ugoda's
Kedowa
Kwanga
Kyondo
Kosova Hills
Ugaya
Kuya R
Kuya
R Gori
Shirati B
Mori B
Mori
Subugu
Loitai
Kitangole
Ft Bukoba
3775
Kibate or Bukora
Mazinga
Semaguta
Nabugabu
Buro
Kagunda
Lukamba
Sewam
Ft Stanley
Masaka
Buru
Kirkoma
Katoma
Kabula
Niskisi
Nsongi
Nabusozu
Kahuro
Katara
Kitangula
Bisharora
Mbarara
L Mburu
Ruezi R
High Hills
Charibika
L Mazingo
Kahega
Kakuto
L Kafungo
L Kachera
L Kiambi
Rakai
Bija
Bwera
Butunzi
Kisozzi
Kasinga
Mpango R
Kitagwenda
Kitasora
Nyangwa
Grant
Raiba
Nabusozu
Kahondo
Mwanu
Kiwogosi
Kirongo
Nyama
Kiriango
Kitumba
Kasozi
Kavera
Kaweri
R Nabukaru
Mugungula
Siboa
Kabutalanga
Chaka
Kiyanga
Kisozzi
Kigoya
Nahuruka
Kitasora
Kisimba
Kiyale
R Misisi
Bawanga
Kavara
Karuma
Kasrogo
Kisalh
4520
Kwera
Magoma
Kibogo
Kiyaga
Kyazanga
Kitaro
Kenya
Busindi
Kitare
Kasuo
Buvabe
Kikubaro
Yandwe
Kibonga
Kikombo
Bale
Kiyumba
Kagura
Kwaxibonda
Lake Mparongoma
Mulasis
Apollo's Ferry
Kagera R
Bugerere
Kiakoge Ferry
Ikumbua
Sibondo
Mkono
Kisiki
Tabingwa
Dobangulu
Sirwanga
Menia
Mutu
Igangu
Napoleon Gulf
Ft Thrustun
Arthur B
Ft Kampala
Mango
Mando
Ngogwe
Buka Bay
Murchison B
Rosebery Channel
Macdonald R
Kerkeley B
Wakoba
4400
Tunga's
Katawui
Mumia's
4400
Kamabula
Nurogare
Maranas
Tumas
Lusumo
Kakamegas
Yala
Waleene
Lwakhakha
Kaimosi
Mbale
Kibisi
Kamasia
Kisumu
Nyando R
Kano Plains
Kibos
Kisian
Kibigori
Kisii
Kisii
Kisii



DISTRIBUTION OF FILARIA PERSTANS AS KNOWN UP TO DATE OCT. 25. 1902.

ALBERT NYANZA
UNYORO
BUGOMA
BUYAGA
BUGANGADZI
BUGUNGU
BUSUJU
BWEKULA
BUDDU
KABULA
ANKOLE
KOKI
BUKEDI
KIMAMA
BURULI
BULEMEZI
BUSOGA
KAGWE
BUVUMA
MAWOKOTA
BUTAMBALA
KIADONDO
SINGO
UGANDA
Kirota
Masindi
Kibero
Kitanwa
Mswa
Kuboko
Hoima
Mpala
Bugoma
Dweru Gulf
Ygusi
Kiambalango
Mwenda's
Mukwenda's
Kitangezi
Mugema's
Kiyanga
Kakunguru
Apollo's Ferry
Mujasis
Kakumbo
Kaguru
Kwanzibondyi
Kibanga
Yandiwe
Kikaburo
Kisoga
Kitaro
Kienya
Busindi
Tabingwa
Sirwanga
Napoleon Gulf
Mando
Dohamanda
Mutt
Iganga
Menia
Kisiki
Sibondo
Mkono
Nahabe
Mbale
Mt. Debasien 9500 9700
Mt. Elgon
Gwas Ngishu 6000-7000 Plateau
Mt. Sirgoi
Suk Mts
Kavirondo Bay
Port Florence
Kisumu
Maragoli Hills
Nandi Escarpment
Nandi Stn
Port Victoria
Ukelos
Yala or Lukos
Mumia's
Katowai
Kimmbula
Tunga's
Wakula
Kampala
Mengo
Gomba
Entebbe
Buganga
Buvu
Inhamba
Ft. Stanley
Nabugabu
Buvuma
Kagando
Mazinga
Kibale or Bukora
Bukoba
Kitangole
Kahega
Kakuto
Rakai
Bija
Masaka
Nabusozi
Kahuro
Kabula
Katara
Kitanguta
Mbarara
L. Mburu
L. Kachera
L. Mazingo
High Hills
Charibika
I. Kafungo
Ruezi R.
R. Kagera
GERMAN EAST AFRICA
VICTORIA NYANZA
Kosova Hills
Ugaya
Kuya R.
Shirati B.
Mori B.
Mori
GERMAN
Subugu
Loito
Kosova
LUMBWA
Kavirondo
Homa
Lusinga I.
Ugowe B.
Kisumu
Port Florence
Kedowa
Kwanza
Kwara
Jadla
Salisbury
L. Gedge
Kateta
LAKE KIOGA
Naika H.
Bwiro
Kubagambura
Wachitwe
Nalo
L. Kwania
Mt. Mahorzi
Kitao
Kosoka
Lango
Nyero
Kagote H.
Berkate
Karita
Turkwel
Siam or Turkwel
R. Sibiel
R. Murona
L. Norman
Sewaw
Mbai
Sore
Kaheru
Gogoro R.
Lake Nyarogoma
Nyanza
Kaheru
Kaheru
Kaheru

DISTRIBUTION OF THE BANANA CULTIVATION AND CLOTHED PEOPLE.

ALBERT NYANZA
VICTORIA NYANZA
BUKEDI
KIMAMA
Kwara
Jaluo
Lango
Kavirondo
Nandi
Lumbwa
Kosoro
Kisii
Ugaya
Kosova Hills
Kavirondo Bay
Port Florence
Kisumu
Maragolia Hills
Nandi Escarpment
Nandi Stn.
Port Victoria
Ukelos
L. Gedge
Salisbury
L. Norman
Mt. Debasien 9500 9700
Mt. Elgon
14200
Gwas Ngishu
6000-7000
Plateau
Mt. Sirgoi
Suk
Mts
Kapte Simba
Kibboss
Elgorini
Nandi
Kakamegas
Lusumo
Nyando
Kedowa
Kano
Kisumu
Port Florence
Kavirondo Bay
Homa
Lusinga I.
Kosova Hills
Ugaya
Kuya R.
R. Gori
Shirati B.
Mori B.
Mori
Subugu
Loita
GERMAN EAST AFRICA
GERMAN
CLOTHED
NAKED
BUGANGADZI
BUYAGA
BUGOMA
BUNYORO
BUSOGA
BULEMEZI
BUSIRO
BUSUJU
BUTAMBALA
MAWOKOTA
BUVUMA
BUDDU
KABULA
BWEKULA
BUGAVAOBEIRE
BURULI
KIAGWE
KIADONDO
SINGO
ENTEBBE
Kampala
Mengo
Napoleon Gulf
Ngogwe Bay
Buka Bay
Buganga
Bwera
Mawokola
Kirkoman
Masaka
Bukakata
Kalungu
Kakunguru
Kagondo
Kibale or Bukoro
Mazinga
Semuguka
Nabugabo
Lubamba
Buvu
Bisoro
Koganda
Kitangole
Bukoba
3775
Kirota
Masindi
Kiberoa
Kitanwa
Muruli
Hoima
Mpala
Mwenda's
Kiambalango
Kasumba
Bawanga
Kitrogo
Karuma
Kiwagosu
Nakabimba
Kirongo
Nyama
Kiriangá
Kahonda
Mwani
Siboa
Kasinga
Mugungutá
Lwekula's
Kaweri
Gomba
Butunzi
Kisozzi
Sekinga
Kirkoma
Katoma
Nabusozu
Kahuro
Nsongi
Kabula
Katara
Bisharora
Kitanguta
Mbarara
L. Mburu
Ruezi R.
High Hills
Charibika
L. Mazingo
Kahega
Kakuto
L. Kafungo
Rakai
L. Kachera
L. Kiambi
Bija
Koki
Ankole
Kitagwenda
Mpanga R.
Chaka
Kasara
Ruizo
Nyangwa
Ft. Grant
Nabuyaga
Kagera
R. Katonga
R. Kafu
R. Lugogo
R. Kitumbu
R. Mayanja
R. Lugaji
R. Sezibwa
R. Bugerere
R. Musita
R. Mabira
R. Bukoba
R. Nabukaro
R. Misisi
Forest
Tubahoro
Kabugurukwa
Kwaxi
Usamba
Kigangara
Kabango
Bukondo
Vimmata
Kiboga
Kigoro
Kisoga
Kiwuya
Kikubiro
Kitara
Kasagu
Buyobe
Busindi
Njoro
Mityana
Mwera
Kabulakalongo
Kabulakiri
Kaduma
Kikengao
Ngalabuzi
Mujasis
Apollo's Ferry
Kibanga
Yandwe
Bale
Kalembo
Kaguru
Kwazibondu
Ikamba
Sibondo
Mkono
Kusiki
Menia
Tabingwa
Sirwanga
Dobangulu
Motto
Igonga
Wakolus
Wakolus Mts.
Tunga's
Katawai
Mumia's 4400
Nyarogare
Tomia's
Martin's
Lusumo
Isukha
Walerie
Lasoos
Yala or Lukos
Yala
Nyando
Kano
Kedowa
Kwando
Nyando
Nyamo
Homa Peke
Kisasalungo
Kuya
Kimsonia
Nyongora
Awuda
Nyiwa
Ugowe B.
Macdonald B.
Kamumbala B.
Thruston B.
Arthur B.
Lubas B.
Channel
Rosebery
Murchison
Kitara
Kitagwenda

4.

ADULT FORMS AND DEVELOPMENTAL FORMS OF THE TRYPANOSOME FOUND IN SLEEPING SICKNESS.

By ALDO CASTELLANI, M.D. (Florence), (Professor of Pathology and Bacteriology, Colombo Medical College).

[Received July 21, 1903.]

[With 2 Plates.]

In previous publications of mine* I stated that in Sleeping Sickness patients I found, in twenty cases out of thirty-four, a trypanosome, which I considered the cause of the disease. My results have been since then amply confirmed by such an authority on trypanosomes as Colonel Bruce. In this note I propose to give some details on the morphology of the adult parasites and their developmental forms.

Appearances of the Adult Trypanosome.

Fresh Preparations.—The parasite has the usual general outline of the other trypanosomes. It has a worm-like shape, and one observes in it one end terminating with a flagellum, the other more or less bluntly conical, though sometimes it may be quite pointed, an undulating membrane and a vacuole. The protoplasm does not appear to have quite a uniform structure, but rather an alveolar one, as described by Plimmer and Bradford in Tryp. Brucei, although apparently far from being so well marked.

At first the parasite moves fairly actively, but, on observing the preparation for some time with the microscope, one sees that the movements by-and-by become more sluggish, until they stop altogether. Frequently the trypanosome stops near a leucocyte, which by degrees engulfs it. In other instances, after having slowed down very much in their movements, the parasites stop far from any leucocytes and disappear suddenly as if they had been dissolved by the liquid. On

* 'Proc. Roy. Soc.,' May, 1903; 'Journal Trop. Med.,' June 1, 1903.

several occasions one sees in fresh preparations trypanosomes with apparently two well-marked flagella; they certainly are parasites in longitudinal division.

Locomotion.—The trypanosome found in Sleeping Sickness, according to my experience, moves always with the so-called posterior end (blunted extremity) in front, whereas the other known trypanosomes move generally with the anterior end (flagellum) in front. Tryp. Gambiense, for instance, according to the description of Dutton, is usually seen progressing with the flagellum (anterior end) in front, unless there is some insurmountable obstruction, when at times it shoots backwards for a short distance with the blunted end in front.

Vitality of the Parasite outside the Body.—The trypanosome may remain alive in fresh preparations of blood rinsed with vaseline for four to six hours. The vitality seems to be longer in the cerebro-spinal fluid, where it may remain alive for fifteen to eighteen hours. At this time the number of the parasites is very much decreased and the movements of the few parasites left are very slow. After twenty hours no parasites are visible. Differences in temperature, at least to a certain degree, do not seem to affect much the vitality of the parasite. For this purpose some tubes of cerebro-spinal fluid were kept at the temperature of the room (28° C.), others incubated at blood heat, and others incubated at 18° C. by means of the cooling incubator. The results were alike for all the tubes, viz., some parasites still alive after eighteen hours, and no more parasites present after twenty hours.

Stained Specimens.—For staining the parasite, Leishman's modification of the Romanowsky's method gives very good results. Staining the trypanosome in this way, the macro-nucleus, micro-nucleus and flagellum appear red, the protoplasm blue, while undulant membrane remains almost unstained.

The nucleus is generally large and of variable shape. It is as a rule situated in the posterior half of the parasite.

The micro-nucleus (Plimmer and Bradford), or centrosoma (Laveran and Mesnil), does not show apparently any structure. It stains red, but of a much more vivid colour than the nucleus. It is situated very near the posterior end of the parasite, and generally outside the vacuole.

The vacuole is oval and of rather large dimensions. It is situated anterior to the micro-nucleus.

The flagellum takes origin apparently from the micro-nucleus, then following the external edge of the undulating membrane, reaches the anterior extremity, where it becomes free. The free portion of the flagellum is usually longer than in other trypanosomes.

The protoplasm does not stain evenly and not very deeply; it does show some chromatic granules.

The total length of the parasite is from 16 to 24 μ. The width is from 2 to 2·5 μ

The adult forms of the parasites are shown in Figs. 1—5.

Atypical Adult Forms. (Figs. 6, 7, 8.)

Besides the adult forms already described, one meets with other rarer forms with different shape. The parasite has lost its slender outline and has become thickened, in fact almost sausage-like. The posterior end is much more rounded, the free portion of the flagellum is shorter. The vacuolum may take greater dimensions. The protoplasm is less well stained. Round the nucleus there may be several points where the chromatine collects. In analogy to what Laveran and Mesnil have observed in Trypanosoma Lewisi, I am inclined to think that these atypical adult forms are parasites which prepare for division. These forms may be observed in the blood as well as in the cerebro-spinal fluid.

Forms in different Stages of Longitudinal Division. (Figs. 9, 10, 11.)

The very first stages of this mode of division are probably represented by the atypical adult forms already described. Then the division begins probably from the micro-nucleus. Forms as represented in figs. 9 and 10 are fairly frequent; the division has gone so far as the micro-nucleus and portion of the intra-body part of the flagellum, but no signs of the division of the nucleus and protoplasm has yet appeared. Fig. 11 shows a trypanosome in which the division is almost complete. The two new parasites are only kept united by the posterior end. These forms may be seen frequently enough in fresh preparations moving about very actively.

Various Types of Development Forms.

Group 1 (Figs. 12, 13, 14).—In the blood taken from the finger, especially during the last stages of the disease, in the blood of the heart, as well as in the cerebro-spinal fluid, I observed frequently special roundish bodies of about 10 to 14 μ in diameter. Their protoplasm is very finely granular, and one can see in it one or more vacuola. These bodies on a very superficial observation might be taken for amœbæ. This idea of course cannot be entertained, because they never emit pseudopoda. These bodies may change very slowly, their general shape passing, for instance, from a round form to an ovoid one. This change of shape takes place in a long time. I could never follow these bodies long enough to see their ultimate transformation. Stained with the Romanowsky-Leishman method, they show several points where the chromatine collects, and, some-

times, very fine flagella. Sometimes there is only one large vacuole, in other instances there may be several vacuola. Similar bodies have been described by Rabinovitsh and Kempner in Trypanosoma Lewisi. Rabinovitsh and Kempner have drawn them without flagella, but they admit now* that possibly some very fine flagella may be present. The interpretation of these bodies is very difficult; they might be degenerative forms, but I am more inclined to consider them developmental forms of the parasite. Doflein thinks that the Rabinovitsh-Kempner bodies observed in Tryp. Lewisi represent perhaps stages of a sexual reproduction.

Group 2. Amœboid forms. (Figs. 15—19.)—These forms were first described by Plimmer and Bradford in Tryp. Brucei. They are very small, their diameter being 5 to 7 μ; the shape is variable. The typical ones are more or less pear-shaped. At the acute extremity there is very frequently a fine short flagellum. A macro-nucleus and a micro-nucleus are present; sometimes they may be divided. Round the macro- and micro-nucleus there is an unstained portion like a vacuolum. The flagellum takes origin apparently from the micro-nucleus. The amœboid forms I have seen only in the cerebro-spinal fluid. Sometimes one observes a large number of these amœboid forms agglomerate together something like a plasmodium formation. (Fig. 22.)

I do not intend to go into the question as to what these bodies really represent; it is well known how far the opinions of Plimmer and Bradford, and Laveran and Mesnil differ on the amœboid forms of Tryp. Brucei, but I think it is interesting to note that identical bodies are seen also in the trypanosome of Sleeping Sickness.

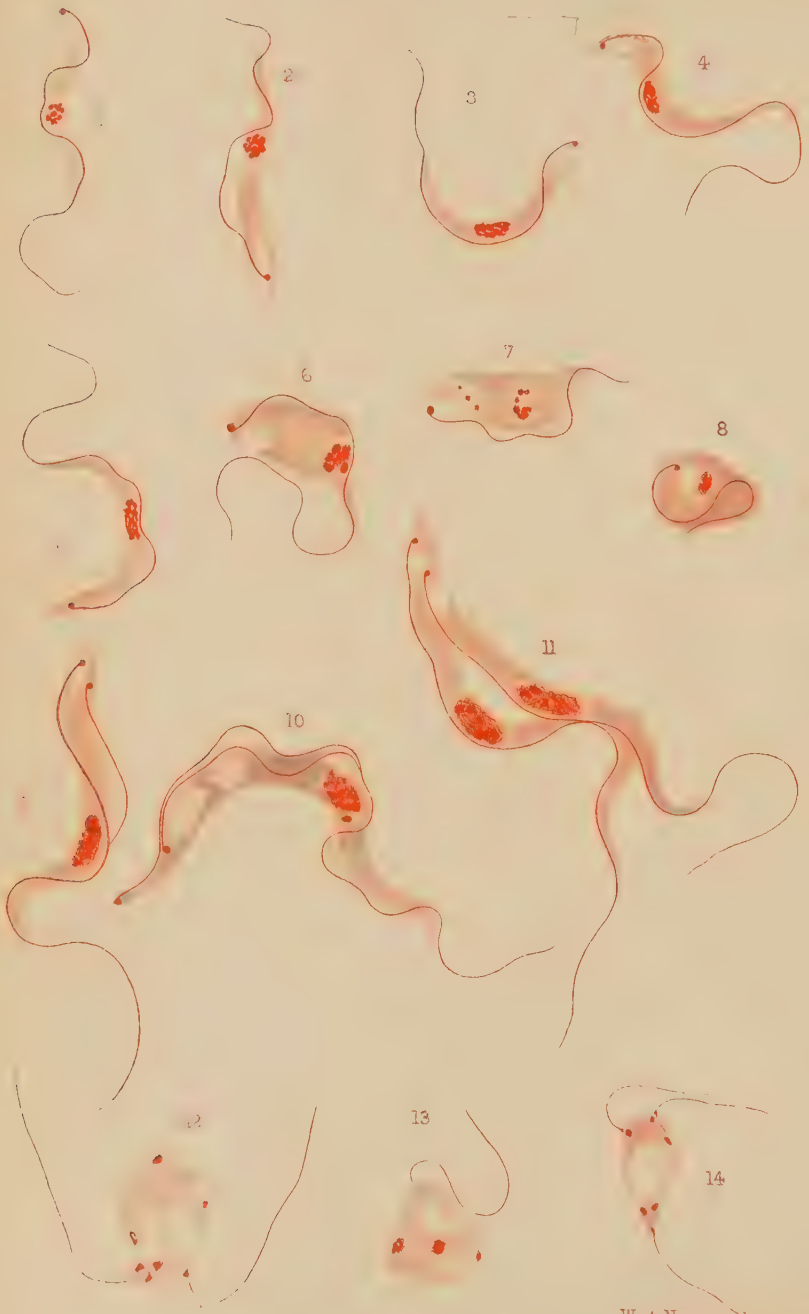
Investigation of Tissues for Trypanosomes.

In animals infected with Nagana or Surra the parasites may be found in some organs, spleen, &c. Very interesting are the experiments of Plimmer and Bradford, who observed the capillaries of brain of animals infected with Nagana full of amœboid forms. Mr. Plimmer has very kindly examined pieces of brain and spleen from Sleeping Sickness patients. So far he has not been able to find any parasites. I hear also that Professor Mott has not yet found parasites in the tissues he has received from Uganda. I have also examined several pieces of tissues of brain and spleen, hardened in alcohol embedded in paraffin and stained with Leishman's method, but so far I have found nothing. Still, too much importance must not be attached to these negative results, as this investigation has been carried out on sections of the tissues. Smears of the tissues might give much better results.

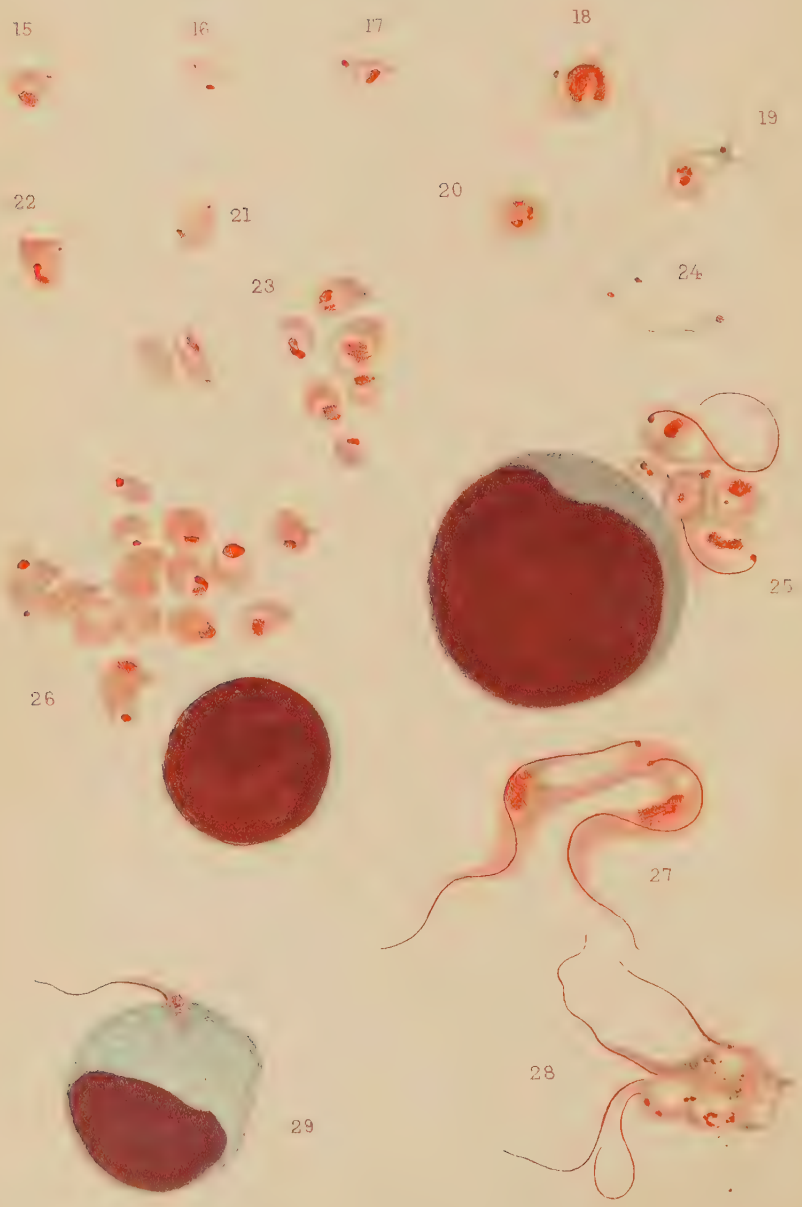
From my researches it would seem that in the trypanosome of

* Oral communication.

Trypanosome of Sleeping Sickness, Pl. 1.



West, Newman chromo.



West, Newman chromo

Sleeping Sickness one must distinguish morphologically several forms: typical adult forms; atypical adult forms; forms in different stages of longitudinal division; Rabinovitsh-Kempner bodies; Plimmer-Bradford amœboid forms. It would be premature to come to any definite conclusions, but from these observations it would seem that in the trypanosome of Sleeping Sickness the multiplication by longitudinal division is not the only mode of reproduction.

DESCRIPTION OF PLATES.

Plate 1.

Figs. 1—5.—Typical adult forms.

Figs. 6, 7, 8.—Atypical adult forms.

Figs. 9, 10, 11.—Forms in various stages of longitudinal division.

Figs. 12, 13, 14.—Different types of developmental forms.

Plate 2.

Figs. 15—19.—Amœboid forms (cerebro-spinal fluid).

Fig. 20.—Aggregation of amœboid forms near a leucocyte (cerebro-spinal fluid).

Fig. 21.—Fusion form? This is probably a degeneration form representing perhaps several trypanosomes which have conglomerated and fused.

Fig. 22.—Aggregation of amœboid forms simulating perhaps a plasmodum formation (cerebro-spinal fluid).

Fig. 23.—Amœboid and fusion forms.

Fig. 24.—Conjugation form? (cerebro-spinal fluid).

Figs. 25—26.—Aggregation of amœboid forms.

Fig. 27.—Conjugation form.

Fig. 28.—Fusion form? This represents, perhaps, the several trypanosomes which have fused together, and are in a state of degeneration.

Fig. 29.—Leucocyte engulfing a trypanosome.

5.

REPORT ON SLEEPING SICKNESS FROM ITS CLINICAL ASPECTS.

By GEORGE C. LOW, M.A., M.B., C.M. (Superintendent London School of Tropical Medicine),

AND

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[Written June, 1903.—Received August 4, 1903.]

The following data were collected in the months of July, August, and September, 1902, in the special hospital erected by the Government, in Entebbe, for the study of Sleeping Sickness.

Synonyms.

This disease is known by many different names:—By the natives of Africa in Uganda as Mongota (To nod). On the Congo as Yela Kwa Tula, Manungina, Lalangolo, N'tansi or N'tola; Nelavane (of the Wolofs), Dadane (of the Sereres), Toruahebue (of the Mendehs).

By European observers it is known as Sleeping Sickness, Sleeping Dropsy, Negro Lethargy, African Lethargy, Die Schlafkrankheit der Neger, Malattia del Sonno, Doenca de Somno, Enfermedad del sueno, Maladie du sommeil, Maladie des dormeurs. It might perhaps be more correctly termed African meningo-encephalitis, or shortly, African Meningitis.

History.

The first to observe this disease was apparently Winterbottom, who wrote a short description of it in a paper entitled "An account of Native Africans in the neighbourhood of Sierra Leone" in 1803. For many years after this little if anything seems to have been written on the subject till 1868, when Dumontier and Santelli both wrote independent papers on the disease. Another paper, by Guerin, appeared

in the following year, but it was not until Corre studied the disease amongst the natives of Senegambia, that a complete and fairly accurate account of this malady was given.

It has been stated that Sleeping Sickness has been seen in Negro slaves imported to the West Indies and in Brazil, but the evidence of this is rather fragmentary, and never appears to have been directly proved. In 1891 Mackenzie published the record of a case which was under his care in the London Hospital, and in 1900 Manson gave a very complete description of two cases in Charing Cross Hospital sent from the Congo by Dr. Grattan Guinness. The pathology of these two cases was very thoroughly worked out by Mott, who first accurately described the pathological histology, and pointed out that the lesion was one of the nature of a meningo-encephalitis. More recently the disease has become more common in different parts of Africa, and much attention has been paid to it in Senegambia by Marchoux and Dantec; in Portuguese West Africa by Cagigal and La Pierre, and also by the Portuguese Commission; in the Congo by Brodin; in Uganda by Cook, Moffat and Hodges.

Geographical Distribution.

The endemic area of Sleeping Sickness is limited to parts of Equatorial Africa.

Until a few years ago the geographical distribution of Sleeping Sickness was limited to West Africa from Senegal in the north to San Paolo de Loanda in the south. It has also been known for many years on the Congo, and lately has spread up that river to beyond Stanley Falls, and it is probably much more common than supposed in hinterlands of most of the West Coast areas between the above-mentioned limits.

On some areas on the Upper and Lower Congo the disease has lately been seen as an epidemic which has killed off large numbers of the population.

In 1900 Cook first discovered that Sleeping Sickness existed in Uganda, and since that date the disease has spread widely, keeping, however, more or less limited to the north shore of the Victoria Nyanza Lake. The epidemic seemed to start in Usoga, and from this centre has spread to Buddu in the west, and down to Kisumu, the rail head in the east. Northwards Cook has recorded cases from Kiadondo, but it does not seem to have spread much in this direction, as it is unknown in some of the northern provinces, in Unyoro and all the districts about the Albert Lake, the same applying to the Nile districts. Its centre of virulence has been in Usoga, in Chagwe, around Entebbe, the seat of the Government, and also in all the islands adjoining the mainland, from the Sese group in the west to the Kavirondo Islands in the east, some of these latter areas having been decimated by this scourge. It has not been

reported in German East Africa to the south and east of the lake, and it is unknown in the Nandi and Masai tribes who inhabit the territories of British East Africa east of Kisumu.

Etiology.

The most different hypotheses have been brought forward to explain the genesis of the disease. Some old observers thought that the disease may have originated from emotional distress connected with negro slavery. Other authors considered the disease a form of sunstroke or a variety of beri-beri or malaria. The disease has also been attributed to scrofula. More recent theories have connected Sleeping Sickness with an intoxication of food, animal parasites, and with bacteria.

Food Intoxication.—Some Portuguese observers, and more recently, Ziemann, have regarded Sleeping Sickness as a sort of intoxication. Ziemann thinks it a manioc-intoxication and compares the disease to Pellagra, which by most authors is also considered an intoxication from food (maize). Against this theory of Ziemann's is the fact that in Uganda Sleeping Sickness has appeared only in the last 3 years, and no change of food whatever has taken place. The staple food of the Baganda is now, as it was before, bananas, and among the tribes near the lake also a little fish.

Parasites. Filaria perstans.—Manson, remarking the singular correspondence between the distribution of the disease and that of *Filaria perstans*, suggested that this parasite might in some way be responsible for Sleeping Sickness. There is no doubt that this theory has some strong points in its favour It would explain well the long incubation period of the disease. Sleeping Sickness, it is said, may develop in negroes many years after they have left the endemic area, and it is well-known that *Filaria perstans* may remain alive for years. This theory has been disproved by the observations of Low, who has shown that in British Guiana, where this parasite is common, there is no Sleeping Sickness, and *vice versâ* in Kavirondo, where the latter disease is rife, there are no filariæ.

Rhabdonema Strongyloides.—The embryo of this worm was considered by Forbes as the cause of Sleeping Sickness. This parasite, according to Tessier, penetrates the mucous membrane of the intestines and reaches the general circulation, and is then retained in the cerebral vessels.

Ankylostoma Duodenale.—Fergusson considers this worm as the cause of the disease. In Uganda this parasite is met with very frequently in Sleeping Sickness, but it cannot be considered the cause of the disease, as the administration of an appropriate anthelmintic frees the patients from it, and sometimes improves the general conditions for a few days, but the fatal course of the disease is not checked.

Bacteria.—Cagigal and Lepierre in 1897 announced that they had isolated a bacillus from the blood of a patient suffering from Sleeping Sickness, which when injected into animals was able to reproduce the disease; but these experiments were not confirmed later by Brault and Lapin. Next, Marchoux, basing his statements on the following observations, is of the opinion that Sleeping Sickness is due to Fränkel's diplococcus. At the *post-mortem* examination in a case of Sleeping Sickness, where the patient was suffering also from pericarditis, he found Fränkel's diplococcus in the exudation of the pericardium. In a second case, complicated by chronic rhinitis and suppuration in the sinus frontalis, the secretion of the nose showed the same micro-organism. Besides these two observations, to which much importance cannot be attached, Marchoux was brought to his hypotheses by having noticed that Sleeping Sickness sometimes developed in people who had suffered from pneumonia. Lately there have been the bacteriological researches of the Portuguese Commission, and those of Dr. Broeden. The Portuguese describe a diplostreptococcus, which they state they have found constantly in the cerebro-spinal fluid at the *post-mortem* examination of their cases. They state that they have frequently found the same organism in patients during life in cerebro-spinal fluid taken by lumbar puncture. According to their description, the characteristic feature of this germ is that it does not grow at all on gelatine and very poorly on the other usual culture media.*

Dr. Broden, of the Bacteriological Laboratory at Leopoldville, describes as the cause of the disease a bacillus which is slightly motile, producing a pellicle on bouillon and growing abundantly on potatoes. He was able to find the germ constantly in the blood of all his patients. The bacillus was not agglutinated by the blood of patients suffering from Sleeping Sickness.

In non-complicated cases Castellani has found frequently a streptococcus. Out of thirty-nine cases, he has grown streptococci from the blood of the heart in thirty-two, and from the liquid of the lateral ventricles in thirty. The germ does not seem to be present frequently in the organs. He has found it a few times in microscopical sections of the brain and spleen. During life he found the germ very rarely, and only in the last stages of the disease. From the blood taken from a vein he grew it once only, although he examined bacteriologically the blood of thirty-seven patients, repeating the investigation several times and with different methods. He examined the cerebro-spinal fluid obtained by lumbar puncture in twenty-eight patients; in five he had positive results, but only a few hours before death, with the exception of one case, in which he found

* In a recent communication the members of the Portuguese Commission have greatly modified the description of their organism, stating that it also grows on gelatine and on the other usual culture-media.

the streptococcus several days before death. In six bacteriological examinations of the urine he grew the germ once; this was in the same case in which it was found in the blood.

In three patients he punctured the spleen during life; the bacteriological examination of the spleen juice, obtained in this way, always gave negative results. The bacteriological examinations of enlarged lymphatic glands, removed during life, was negative.

Trypanosoma.—Since November, 1902, when he began to use a special technique, Castellani observed very frequently a trypanosoma in Sleeping Sickness patients. He found this trypanosoma in the cerebro-spinal fluid of twenty out of thirty-four patients. In the blood, among the few cases examined, he found it in one, and in several cases he observed special bodies which he considers developmental forms of the parasite. These bodies can be seen also in the cerebro-spinal fluid. In this fluid may be present also other bodies very small, 4—6 μ in diameter, which, when stained with Romanowsky's method, show a macro-nucleus and a micro-nucleus, from which a flagellum takes origin. These bodies are identical with the amœboid forms described by Plimmer and Bradford in Tryp. Brucei.

Predisposing Causes.

So far, the disease has been observed in negroes only. A case was observed by Chassaniol in a mulatto, and one by Clarke in a creole boy. It is said to occur in negroes long after they have left the endemic area. Sex does not exercise any influence, neither does age. The disease is equally frequent among children, young and old people. As important predisposing causes, Clarke mentions disorders of circulation, mental depression, bad and insufficient food. It may also be that parasites like filaria perstans, ankylostoma duodenale, etc., so common in natives, impairing the natural forces of resistance, may play a certain rôle as predisposing causes in the ætiology of the disease.

Symptoms and Clinical Features of Sleeping Sickness.

General Sketch.

The symptoms of the disease begin very insidiously, some slight change in the former mental attitude of the patient being the first thing noticed by the relatives of the patient. Next a disinclination to work, with a tendency to sit about and rest more than usual, appears, and at this time headaches and other transient pains may be complained of, especially pains in the upper part of the chest. The facial aspect now also changes and a previously happy and intelligent looking negro becomes instead, dull, heavy and apathetic. Once those changes have appeared, the disease may run an acute or more or less chronic

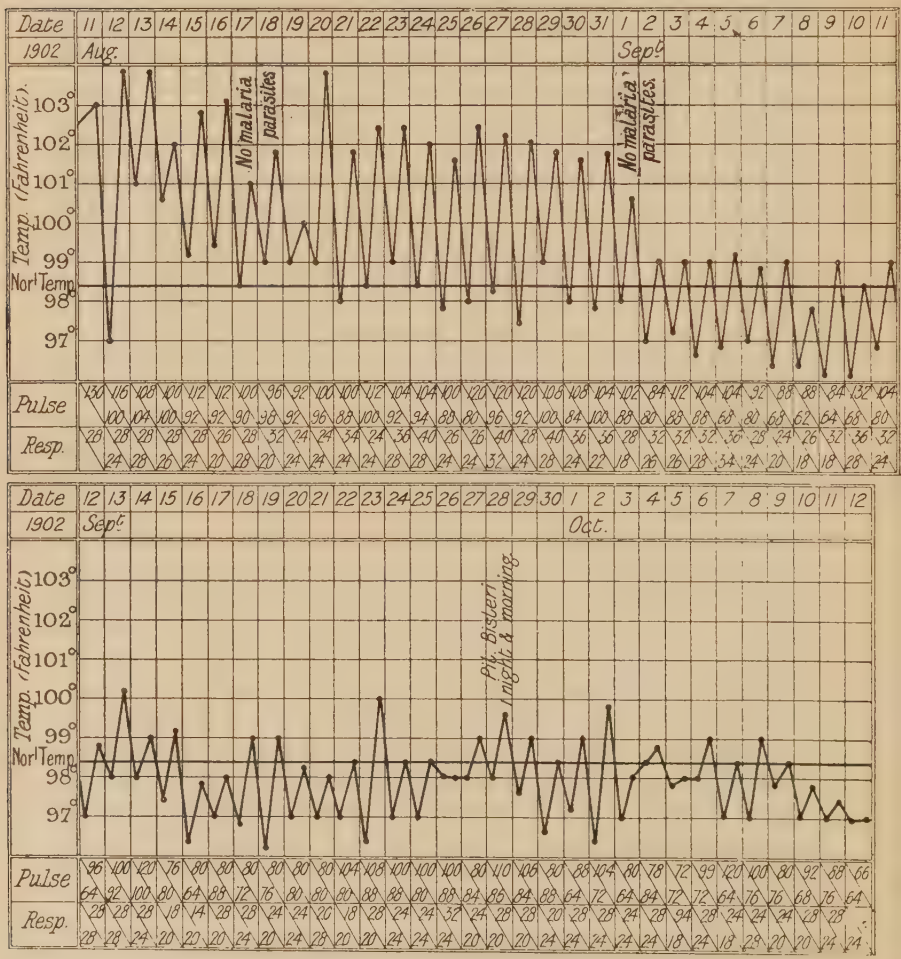
course, progressing however to its ultimate fatal termination. It is about this time that one usually sees the case, and an ordinary inspection will reveal many of the following points:—There is the dull, heavy, stupid look, a slowness in answering questions, and when speech does come it is often mumbling, slow and thick; the gait is best expressed by the term shuffling. Headaches, vague pains and chest pains may be complained of. The tongue may or may not at this time show the characteristic fine tremor, and in some cases this may also be noticeable in the hands. The skin is often soft and smooth, or it may be slightly roughened. Glandular enlargements common amongst all natives may be prominent, but in some cases this may be very slight. The temperature—a very important point—is elevated, rising in the evenings to 101° or 102° F., falling to subnormal in the morning, the range often extending over 4° or more, and the pulse of very low tension is accelerated, varying from 90—130 beats per minute. These two symptoms are of the greatest diagnostic importance in the early recognition of the disease. On interrupting the examination and quietly watching the patient, he will probably sit down, his head may nod, his eyes close, and he remains in this drowsy lethargic condition until again asked some question. If one take such a case into hospital, for the first few days a slight improvement may take place; the patient gets up from his bed daily, sits about the doors of the hospital, sometimes walks about outside and takes a little more interest in life, especially at meal times. Soon, however, depending on whether the disease is to run an acute or chronic course, the individual gets worse, he stays in bed more, becomes more drowsy and lethargic, though not actually sleeping, walking at the same time becomes more difficult, and he eventually remains constantly in bed.

Tremors now usually become marked, these being of a fine nature. They are best seen in the tongue and arms. The skin may become rough and lose its lustre, but eruptions, though they have been described, are not common. Emaciation and general weakness becomes pronounced; the knee reflexes, which were at first somewhat exaggerated, become diminished, the motions are passed involuntarily in bed, and saliva often dribbles from the mouth. Drowsiness, which has gradually been increasing, now passes on to coma, from which the patients can only be roused with difficulty; the temperature falls to subnormal, in rare cases convulsive fits appear, and the patient dies in a complete state of coma. This is the common course of an ordinary acute case of the disease, the different changes taking about a month or six weeks for completion. In the chronic cases the symptoms develop more slowly, and they remain more constant for considerable periods of time without any advance, but ultimately the patients pass into the later stages described above and eventually die.

Analysis of Symptoms.

The Temperature. Regular Course.—The course of the temperature is marked in typical cases by an evening rise and a morning fall, the range often extending over 4° or more. The night temperature varies from 101°, 102° to 103° F., and the morning temperature is generally about the normal, sometimes, however, a little over this or often distinctly subnormal.

Charts 1 to 5.—Typical Temperature Charts.



Pil. Bisleri = Iron, arsenic and quinine.

Chart 2.

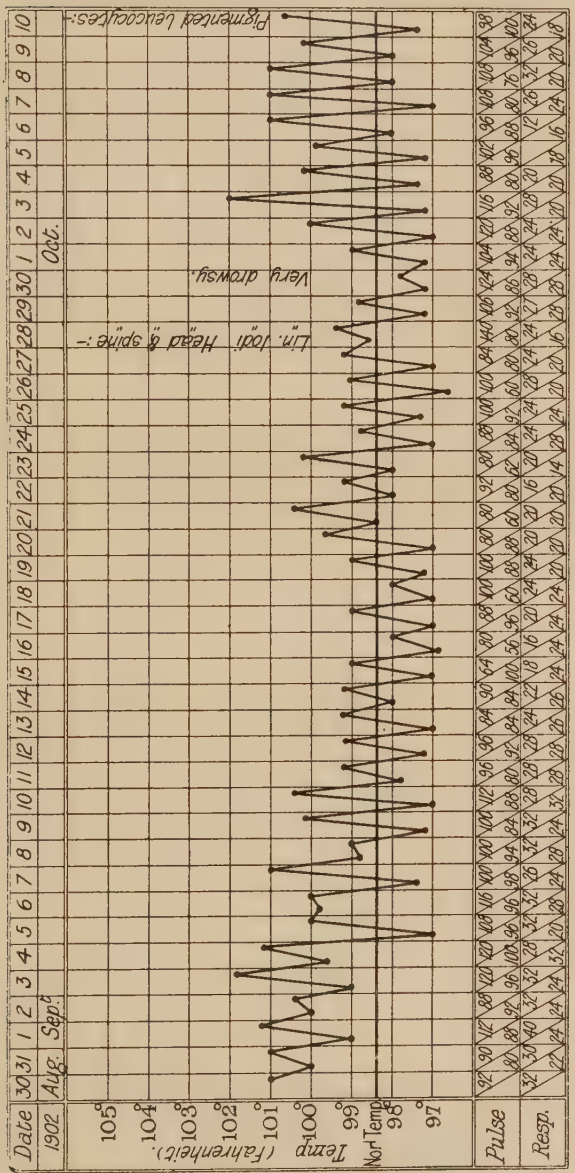


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Chart 3.

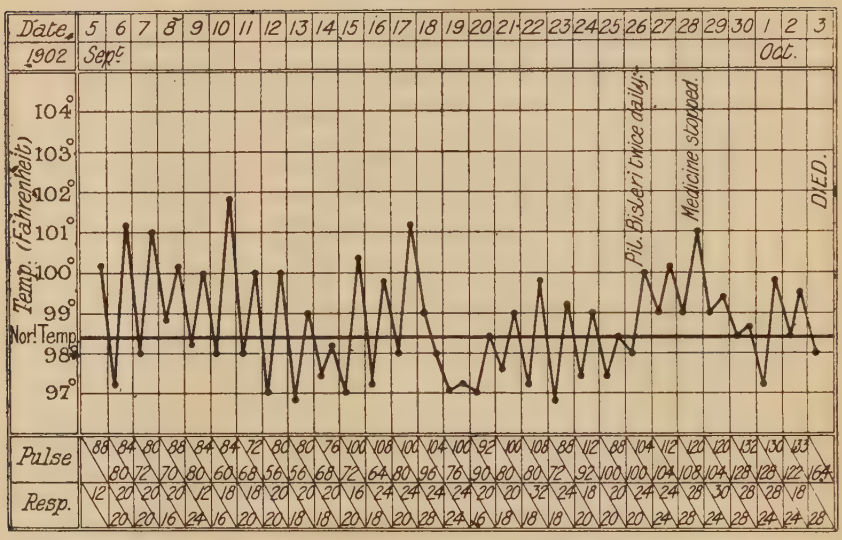


Chart 4.

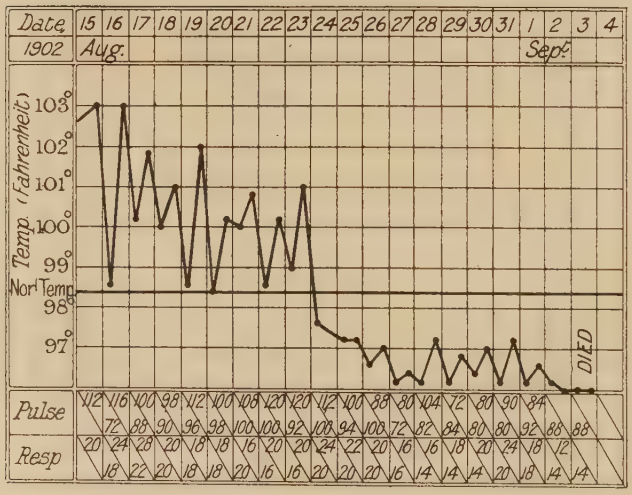
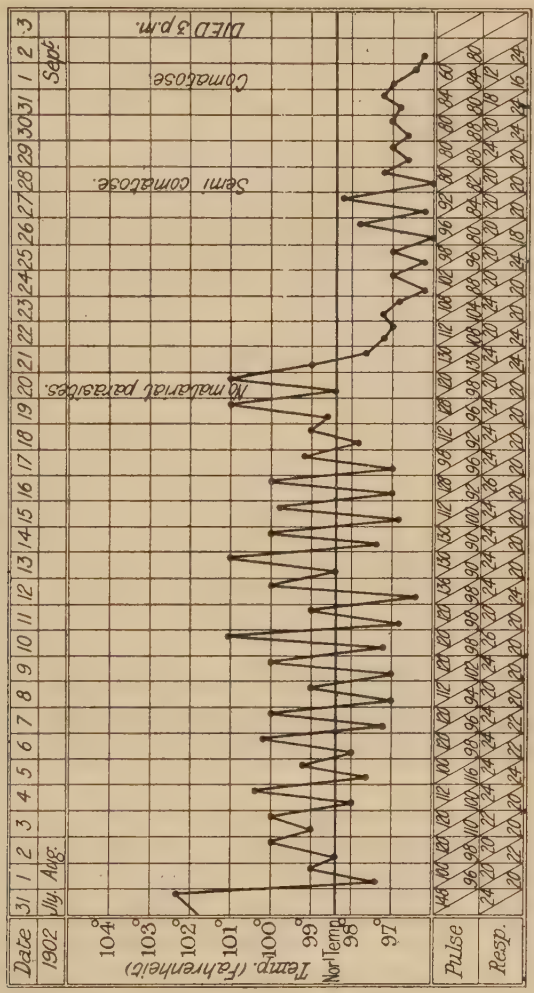


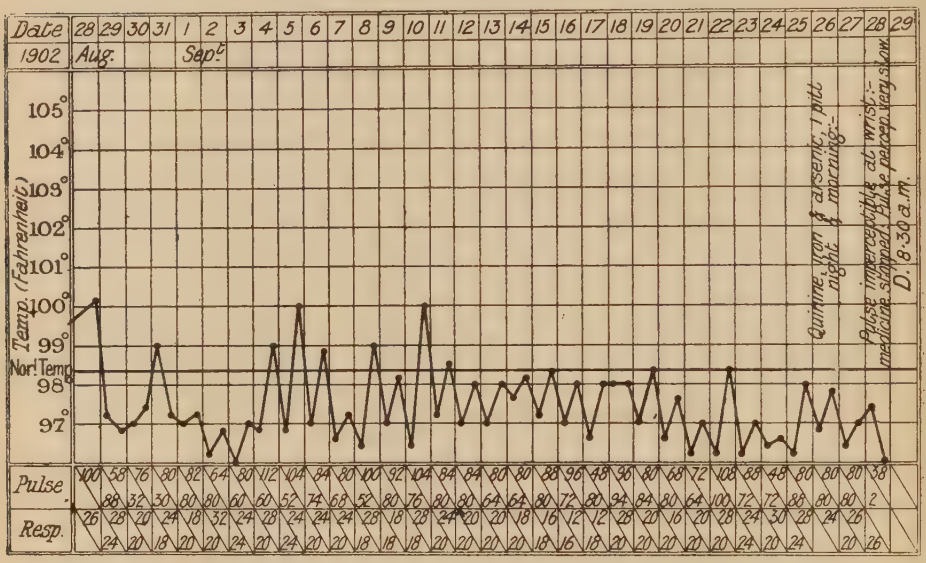
Chart 5.



This regular type of temperature may go on for three or four weeks, and then it may become distinctly irregular, sometimes almost keeping normal, with the exception of a slight evening rise now and again; this condition persists for a variable time. or it may again become regular. Just before death, one week or two weeks before, the temperature almost always becomes subnormal and remains so during the whole day. This latter condition warns one that the end is near. The rise of temperature is not accompanied by any special subjective symptoms, there is no rigor and no sweating, and it is not uncommon to see a patient with pyrexia of 103° F. walking about apparently with little the matter.

Variations from the typical temperature curves are from time to time met with, the chart, below, with the exception of one or two evening rises, showing a practically subnormal temperature throughout the course of the disease; the case, however, was an acute one, and ended quickly in death.

Chart 6.—Atypical Temperature Chart.



In another type of case the temperature may remain high for several days without showing any marked morning remission. As a rule, however, after a few days it reverts to the usual type—no cases were ever seen by us without pyrexia at some part or other of the course of the disease. The ordinary temperature curve may be modified by intercurrent diseases, the most frequent being malarial fever; here the maximum is unusually high, often reaching 103° F. or over, and the remissions are slight, sometimes never coming below 100° F. Parasites may then be demonstrated in the blood, and the administration of quinine causes the temperature again to revert to its ordinary course.

The Circulatory System.

The study of the pulse in Sleeping Sickness is very important, especially in the early diagnosis of the disease.

The frequency is very rarely below normal; it is usually of a rate of 90—130 per minute, but great variations may be got at different times of the same day. The ratio to the temperature is inconstant, a very high frequency often being associated with a low temperature, but

in the last stages a low frequency associated with the subnormal temperature is the rule.

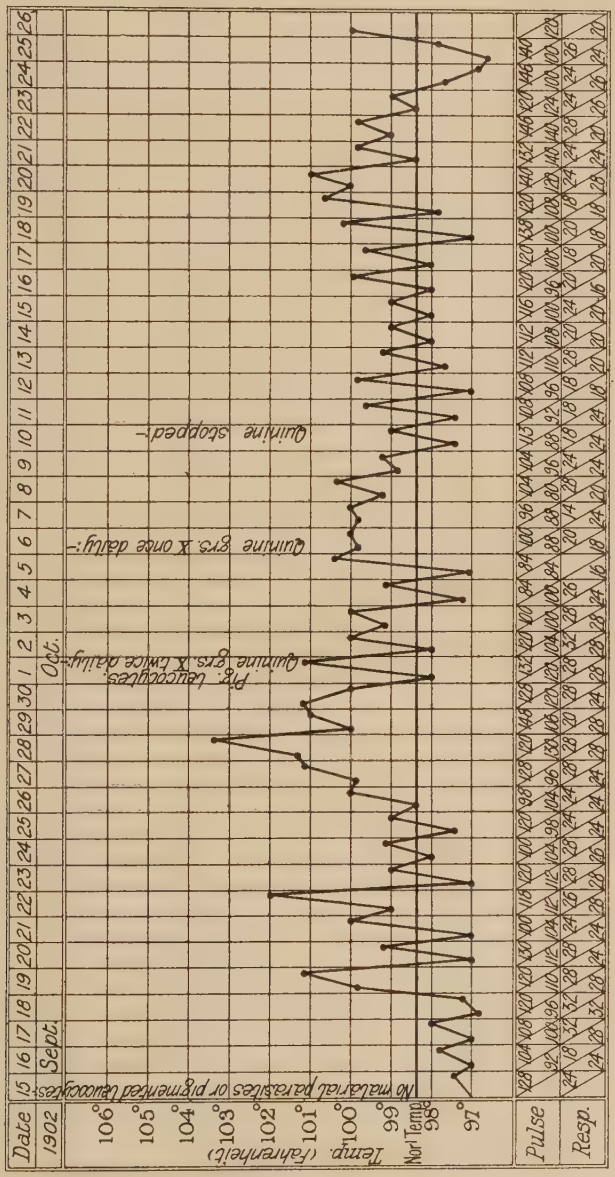


Chart 7.—Malarial Complication.

The rhythm is almost always regular in time and equal in force till the last stages, when some of the beats may be weaker than others; just before death, or even two days before, the pulse becomes imper-

ceptible at the wrist. Dicrotism is uncommon, but it may be present when the fever is very high.

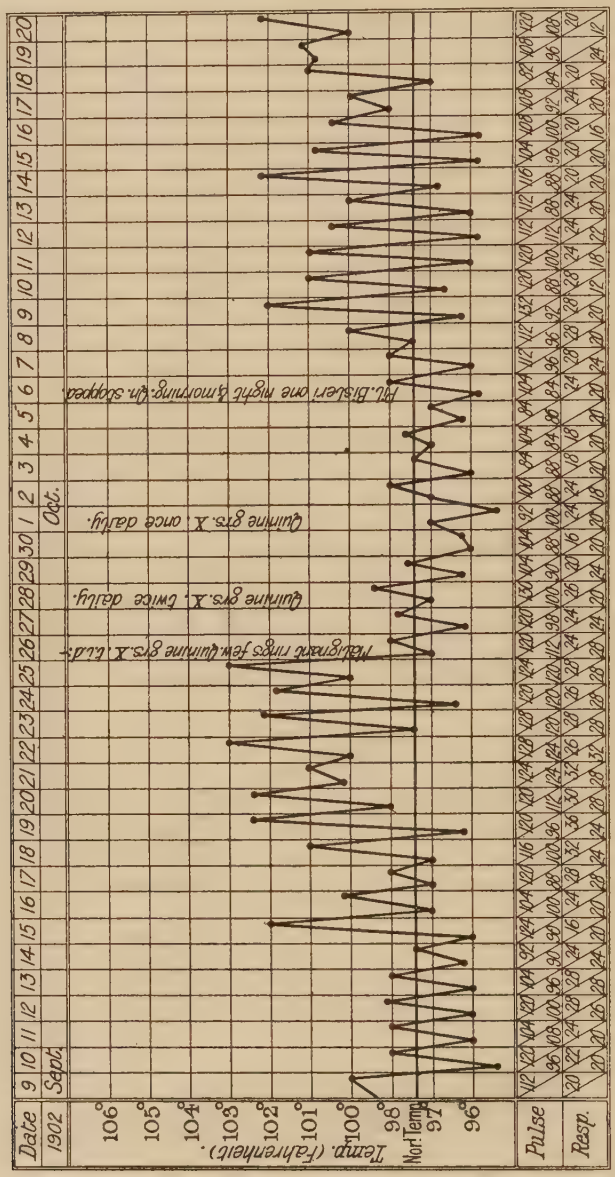


Chart 8.—Malarial Complication.

The volume is always very small, and is associated with a remarkably low tension, the vessel wall exhibiting no thickening or other abnormalities in uncomplicated cases.

There are no subjective symptoms as regards the heart; palpitation, cardiac pain, dyspnœa and giddiness are all absent; the physical examination shows little abnormal with the exception of the increased rhythm, and in some instances an increase in the transverse diameter. Inorganic bruits may be present, but neither valvular diseases nor endocarditis ever complicated any of the cases.

The Respiratory System.

The respirations, regular in time and equal in force, are always increased, especially so in the evening; they correspond fairly closely to the increased pulse rate, and their number per minute generally is between 20 and 30. In the last stages of the disease Cheyne-Stokes breathing is common.

The physical examination of the respiratory system in the first stage reveals nothing. In the later stages some congestion and œdema of the bases of the lungs, due no doubt to the recumbent posture in bed, are commonly met with, and pneumonic patches may appear.

Alimentary System.

In the early cases there is not much to be noticed in this system. In some patients the appetite appears to be slightly increased, and, even in the advanced cases, when completely bedridden, nourishment is still readily taken. Dyspeptic symptoms are as a rule absent, and vomiting is never seen. The bowels are constipated. Herpetic eruptions are never seen on the lips; ulcerative stomatitis in a slight degree is sometimes seen, and the gums in rare cases become swollen and spongy. The tongue is flabby and covered with a dirty fur; the papillæ on the dorsum may be prominent, and, in the last days of life, sordes tend to accumulate in the mouth. The breath often has an offensive odour. An inspection of the abdomen often reveals a certain degree of bulging; the stomach is generally confined to its normal limits, though a certain amount of dilatation may be present, due no doubt to the large amount of vegetable food eaten. The spleen is always enlarged, hard, and not tender, but the question of antecedent malaria in producing this must always be considered. The same may be said of the liver, though the degrees of enlargement of this organ are not so marked as those of the spleen. Pressure on the abdomen never elicits pain; the colon, especially in emaciated individuals, may often be felt full of hard fæces.

Integumentary System.

Roughness of the skin, which has been considered so diagnostic of this disease, is not, according to our experience, by any means a

constant feature, many cases exhibiting a perfectly smooth skin up to the day of their death. The same must be said of the eruptions, which are not frequent, and the type of eruption when existing is similar to that seen very commonly amongst the healthy natives. The most common eruption is a papulo-pustular one; the lesions are most frequently seen on the dorsum of the hands, extensor aspect of forearms and on the back. The pustules are generally isolated and have no tendency to run together. Pruritus often accompanies this condition, and the skin, as in other marasmic conditions, may, especially in the chronic cases, become rough, scaly, and lose its lustre. Forms of eczema and ordinary scabies are sometimes seen.

Lymphatic System.

Enlarged glands in most descriptions of this disease have had special stress laid upon them. A special study of many natives, healthy or suffering from other ailments, shows at once that this condition is a common one, and cannot therefore be regarded as a special symptom of Sleeping Sickness. What produces this polyadenitis is difficult to determine, as it occurs in young children and also in adults. Perhaps frequent skin diseases, syphilis, and verminous invasions may be responsible. These enlarged glands are most commonly found in the posterior and anterior triangles of the neck, the submental region, the submaxillary region, Scarpa's triangles, and in the groins. In emaciated individuals the abdominal ones can also be felt. Their size varies from a small bean to a hazel-nut, and they are always hard and firm in consistence. They never become adherent or cause ulceration of the skin. The condition generally is a chronic one, though in some rare instances suppuration may be seen.

Nervous System.

The dull apathetic look is one of the most characteristic features of the disease, though far from being a constant one. The expression is heavy, and shows very little emotion. There seems to be a slight loss of intelligence, but memory is not impaired. When spoken to they respond after a considerable interval. Speech is not, however, specially affected, there being no stammering nor slurring in the production of the individual syllables.

Proper sleep is not a symptom of the disease as is so generally supposed; a patient if left alone is often seen to nod his head and close his eyes, but the slightest touch, such as stroking him, or any noise, will make him open his eyes at once. The total amount of sleep may be above the average in a few cases, but the usual feature is one of lethargy, indifference, and drowsiness, and even this in several cases is not well marked, while in others it may be entirely absent.

Maniacal attacks sometimes usher in the onset of the disease. Headache, chiefly occipital, indefinite pains in the chest and sometimes in the joints, especially in the knees and ankles, are sometimes complained of.

Sensory Functions.—These at first are generally normal, though even early there may be some hyperæsthesia at the trigeminal points. Touching or moving the patient, especially in those cases where flexure contraction or rigidity of the muscles of the neck is marked, causes pain, evidenced by the patient crying out. General or local anæsthesias were never observed. The temperature sense is unimpaired, and the same seems to be the case with the muscular sense, though this is difficult to be sure of in dealing with savages.

Motor Functions.—Especially towards the end of the illness, when emaciation may be marked, the muscles become frequently wasted and flabby, though in the acute cases death often happens without much loss of the muscular nutrition. The motor-power, at first powerful, diminishes towards the end. In many cases a certain degree of incoordination is distinct, and in a few cases Romberg's symptom is present. "The gait is a typical one and best described by the term 'shuffling,'" the feet not being raised from the ground, but being pushed forward.

Abnormal muscular movements are perhaps the most striking feature of the disease; fine tremor in the tongue is very constant, and a somewhat coarser tremor is also usual in the hands and arms, any purposeful movements, as lifting a cup to the lips, often increasing it. In a few instances tremor is found in the legs and muscles of the trunk so excessive as to cause shaking of the bed in which the patient lies. In a few rare cases, which were ultimately proved at the necropsy to be undoubtedly Sleeping Sickness, no tremors were visible during the whole course of the disease.

In the last stages rigidity is common in the muscles of the neck and flexure contractions of the legs on thighs, and the thighs on the abdomen may at this time be extreme. Fits of an epileptic nature sometimes occur, either general or localised to a group of muscles.

Paralysis is rare; it was once noticed in the muscles of the right side of the face, and in another case in one arm. Hemiplegia or paraplegia were never seen, and choreic movements were never observed.

Reflex Functions.—The superficial reflexes are generally normal. The deep, exaggerated at first, afterwards become lost. There is never clonus. The organic reflexes are normal, but during the last weeks the motions are passed involuntarily, this, no doubt, depending on weakness and inability to get out of bed rather than on any definite paralysis.

Babinsky's symptom is never present.

Lumbar puncture was performed in the great majority of cases.

The cerebro-spinal fluid escapes generally with increased pressure, though in a few cases the pressure may be normal or even decreased. The fluid appears as a rule perfectly clear and colourless. In some few cases it is slightly turbid. Albumen is found only in traces. The liquid always reduced Fehling's solution. The leucocytic formula is mononuclear.

Special Senses: Eyes.—The pupils are equal and as a rule dilated. They contract to light and during accommodation, though in some cases both reactions are sluggish. There is never nystagmus nor any ocular paralysis. (Hearing, taste, and smell are, apparently, unimpaired.) There is no change in the fundus or retina.

Urinary System.

As has been mentioned before, weakness of the sphincter vesicæ has been frequently met with in the last stages of the disease. We have never observed retention, nor any temporary difficulty in starting micturition. The amount of urine passed during the 24 hours is generally normal at first, while in advanced cases a distinct increase of the secretion may be noticed. The urine when freshly passed is generally clear, and of a very pale colour. It is of a low specific gravity and does not give any characteristic odour. The reaction is almost constantly alkaline, this probably being due to the vegetarian diet of the patients. Carbonates are abundant and also earthy phosphates. The amount of urea excreted is below the average. Albumen is absent as a rule, though a slight trace of it may be present sometimes, when the pyrexia reaches a high degree. Sugar is always absent, and so are bile pigments in uncomplicated cases. The sediment is chiefly composed of oxalates, triple phosphates, and carbonates.

Sexual System.

At first sexual desire is apparently not decreased, but later it is lost as the sexual system partakes of the general nervous debility. Menstruation may remain normal for a long time, but disappears as a rule in advanced cases. In the mucous secretion of the vagina, which is frequently increased and of acid reaction, one often finds in several cases a form of flagellatum with three flagella and an undulating membrane, showing very active movements (Trichomonas vaginalis) (Dormé). The same flagellation was observed also in the vaginal secretion of apparently healthy women.

Fœces.

Constipation, as already mentioned, is very frequent. The stools are generally hard, of greenish colour, and almost free from odour. Portions of undigested food, large shreds of vegetable tissues, and long fibres are

often to be seen. This appearance of the fæces depends certainly on the diet of the patients, which consist practically of vegetables only. Microscopically there are innumerable micro-organisms, epithelial cells, crystals of triple phosphate, oxalate of lime, and occasionally Charcot's crystals; there are also many vegetable cells, starch granules, and frequently yeast fungi. Ova of different parasites are always present. The commonest of these are Ankylostoma duodenale and Ascaris lumbricoides; less common, Trichocephalus dispar. In two cases Anguillula intestinale was observed. In several instances ova of Bilharzia hæmatobia were present in the fæces. The ova had the characteristic spine, not at one pole but a little to one side. Some had no spine at all; free embryos were also observed. Among the protozoa Trichomonas intestinalis is very common.

Hæmopoietic System.

Anæmia in varying amount is constant: the average number of the red blood corpuscles per c.mm. being 3,500,000 or thereabout. So many other blood-destroying factors are present, such as malaria and ankylostomiasis, that it is difficult to say what part they also play in producing this condition of the blood. In some cases if cyanosis exists just before death there may be an abnormal increase in the R.B.C., one such case showing 6,200,000 per c.mm. on the day of death. Commoner than this, however, is a gradual fall to 2,000,000 or under. The hæmoglobin is generally reduced in relationship to the amount of the anæmia. The leucocytic count shows no absolute increase from the normal until just before death, when a certain number of the cases get a well-marked terminal polymorphonuclear leucocytosis. Relatively the large mononuclear elements are increased; but malaria must again be taken into account in considering this. Especially in young subjects a relative increase in the number of eosinophile leucocytes is often met with; this is probaby due to helminthiasis.

The main features of the blood may be summed up as follows: An anæmia of varying degrees, with a relative increase of the large mononuclear leucocytes, malarial parasites and pigmented leucocytes, indicating malarial complications, are frequently met with.

Complications.

Bed sores, when emaciation is extreme, are common, especially so when one considers the nature of the beds in which the patients are treated. With ordinary care they may be largely avoided. They are most apt to be got in the chronic type of cases. As already stated, under the integumentary system skin affections of varying nature are common. In a few cases boils of a pemphigoid nature were observed,

the distribution of these being limited to a few spots. Ulcerations of the hands and feet due to chiggers are common.

Epistaxis is rare. Different forms of laryngitis were met with, the cause of death in one case being œdema of the glottis. Bronchitis, œdema, and nasal congestion are all common in the last stages. Lobar pneumonia is one of the most frequent causes of death. In children and old people broncho-pneumonia is common.

Valvular diseases of the heart and affections of the kidneys are extremely rare.

Sometimes pseudo-dysenteric symptoms, due to the presence of bilharzia parasites, were observed.

Course of the Disease.

The incubation period is an uncertain one, and there is the greatest difficulty in determining this point, because of the very insidious onset of the disease. The rapid spread of the disorder in Kavirondo would seem to show that the incubation period may be a more rapid one than is generally supposed. After the stage of incubation is over the symptoms, as stated in the general sketch, begin very insidiously, and the course of the disease is a very variable one. The average duration is from 4 to 8 months, and not infrequently cases much more acute than these are seen. Very chronic cases, running a course of more than a year's duration, are very rare.

The disease clinically may be divided into three stages. The first stage is represented by somewhat indefinite symptoms, vague pains, feelings of indisposition, and sometimes a slight degree of drowsiness. Fever is always present (an important point). The second stage may be designated as the stage of tremor. With an increase of the former symptoms, tremors accompanied by other nervous symptoms appear. The third stage is characterised by intense weakness. The patients now become bedridden, they loose control of their sphincters, a coma which progressively deepens appears, temperature falls to subnormal, and the patient passes away.

Pathological Anatomy.

The pathological changes as pointed out by Mott are essentially those of a chronic meningo-encephalitis, and a meningo-myelitis. Macroscopically in the large majority of the cases there is not much to be seen. The calvarium is not thickened. The dura-mater shows little change; it is seldom adherent to the skull, but may be to the subjacent arachnoid. The sub-dural fluid is not usually in excess, though some cases show a marked increase. The pia-arachnoid, generally clear, is, however, sometimes opaque and slightly thickened. In some instances it is adherent to the brain, and on being stripped off causes some erosion;

the vessels in this membrane may show some congestion. The subarachnoid fluid is usually pale straw coloured and is often in excess. In a few cases it is slighty turbid, and in two instances was distinctly purulent, especially over the sulci and round the vessels in the membranes. Recent lymph in small amount may be found. On section the substance of the brain is moderately firm and nothing abnormal is to be seen, with the exception sometimes of excess of fluid in, and dilatation of, the lateral ventricles. The pituitary body, the pons cerebellum and medulla oblongata show no macroscopical changes. The membranes covering the spinal cord show the same appearance as that of the brain, and sections of different areas reveal nothing abnormal macroscopically.

The other organs also show little change. The heart is usually flabby, pale, and may show signs of myocardial changes. Congestion and œdema of the lungs are almost always seen. Patches of bronchial pneumonia are common. Pleural effusion was never observed, though in some cases pleuritic adhesions were noted.

The livers in all the series of cases examined showed distinctly the appearance of chronic malaria, and the same was also found in the spleen, which was often considerably enlarged. Section of the spleen showed the characteristic pigmentation of malaria. The kidneys, suprarenal capsules, and pancreas were in all cases normal. The stomach, usually of an ordinary size, may sometimes be dilated, and may show a patchy congestion, the intestines often exhibiting a similar congestion. Parasitic nematodes, *Ankylostoma duodenale*, and *Ascaris lumbricoides*, were invariably present. The large intestines in four cases exhibited marked congestion and superficial erosions of the mucosa due to the ova of Bilharzia hæmatobia. *Trichocephalus dispar* was always found in the cæcum. In the mesentery the adult forms of *Filaria perstans* were often found in the connective tissues, and bilharzia adults in the mesenteric veins.

Enlarged glands in all cases were a general feature; in the superficial areas the size varied from a bean to an almond, but the retro-peritoneal ones were often much larger, sometimes reaching the size of a walnut. This condition, however, was met with also at the autopsy of cases suffering from other diseases. The bladder, urinary and sexual organs were never affected.

Microscopically definite and characteristic changes are always to be met with in the nervous system. A mononuclear leucocytic infiltration is always seen on the meningeal surface of the brain spreading down into the sulci, and affecting in varying degrees the perivascular spaces around the blood vessels in the substance of the cerebrum pons, medulla and spinal cord. The leucocytic formula of the cerebro-spinal fluid is also mononuclear.

The degree of perivascular infiltration varies considerably and does

not follow a corresponding ratio to the symptoms. The nerve cells show frequent pathological changes, being often altered and irregular, very much resembling those described by Mott. The tangential and inter-radial association fibres are diminished in number, and many show great atrophy. Sections stained by Loeffler's and Gram's methods never showed micro-organisms, with the exception of four cases in which cocci which retained the Gram's stain were seen in couples and short chains.

The pia-arachoid of the spinal cord showed a similar leucocytic infiltration, which was also seen in varying degrees around the vessels in the substance of the cord. The axis cylinders and medullary sheaths of the nerves in the cord (stained by Marchi's method) may show a diffuse degeneration in some cases.

The arteries both in the brain and spinal cord never exhibited signs of endarteritis. The peripheral nerves (sciatic, anterior and posterior, tibial, ulnar and radial, etc.) showed no signs of degeneration, with the exception of deposits of fat in the epineurium and sometimes in the endoneurium. As regards the other organs of the body there is not much to note: a leucocytic infiltration may be seen in almost all the organs, and especially in the heart, and secondary changes such as occur in infectious diseases may also be got, namely, cloudy swelling of the liver cells and degenerative changes in the myocardium.

The liver and spleen of all the cases examined in the *post-mortem* room in Entebbe showed chronic malarial changes, and plenty of recent or old malarial pigment. Sections of the spleen stained for micro-organisms showed in very few cases the same cocci as described in the brain. Sections of the bilharzial erosions showed a marked hæmorrhagic condition of the mucous membrane, and the presence of many typical ova. No infections of the bladder by bilharzia were seen, and it would thus appear that rectal bilharzial disease is the common form in Uganda, the ova in all such cases being furnished with a lateral spine.

Diagnosis.

The diagnosis in early cases may be exceedingly difficult, as the typical features of the disease (tremor, etc.) are generally absent. The most important fact on which to base the diagnosis in this stage is the evening rise of temperature and the increased rate of the pulse. Later on, when the definite symptoms appear, there is little difficulty, as the disease does not resemble closely any of the common nervous disorders, with, perhaps, according to Mauther, the exception of Wernicke's acute poliencephalitis superior.

Beri-Beri.

Sleeping Sickness has been considered by some authors to be a form of Beri-Beri. To anyone with experience of the two diseases such a

mistake should never occur. The latter disease is a peripheral neuritis which comes on rapidly. In the wet form there is marked œdema, in the dry form, wasting of the muscles. In both forms the knee-reflex is abolished, and hyperæsthesia of the muscles is a prominent feature. In Sleeping Sickness these symptoms are absent, and the tremor, the pyrexia and the lethargy at once distinguish the two diseases.

Intracranial Syphilis and Tumours.—Some cases of intracranial syphilis and tumours of the brain have been described, with a curious tendency to somnolence, so that the person drops off to sleep while at work. Such cases if occurring in the endemic area might present difficulties, but in the former other evidences of syphilis would be present, and in the latter the usual definite symptoms of a cerebral tumour would be found. The temperature which is such a constant symptom of Sleeping Sickness would greatly clear the diagnosis.

Chronic Nephritis.—Chronic nephritis with uræmic symptoms may present some superficial resemblance to Sleeping Sickness. Such a case was sent to our hospital in Entebbe, with the diagnosis of Sleeping Sickness, but a careful examination of the case revealed the fact that the patient's urine contained albumin, and that there was also a well marked albuminuric retinitis. At the autopsy a few weeks afterwards the pathological examination upheld the diagnosis.

Tabes and General Paralysis of the Insane.—In some rare cases the pupils may react to light very sluggishly, and if Romberg's symptom and the absence of the knee jerk also be present, the question of tabes dorsalis must be considered. Strangely enough, though syphilis is prevalent in Uganda, parasyphilitic diseases, according to Cook and to our own experience, are excessively rare. Tremor and pyrexia will lead one to the diagnosis of Sleeping Sickness. Perhaps the disease which most closely resembles Sleeping Sickness is general paralysis of the insane, as in the former disease symptoms of insanity are not infrequently seen. The regular pyrexia almost constantly present in Sleeping Sickness is the best test to differentiate the two diseases.

Prognosis.

The prognosis is a grave one, as the disease always terminates fatally. All the cases in our hospital in Entebbe died, and we were never able to satisfy ourselves as to rumours of people recovering. A native doctor claimed to have cured several patients by incisions over the temples, but the French Missionaries told us that all those patients, after a short apparent improvement, died. The disease appears to be invariably fatal.

Treatment.

Many drugs were tried without any definite results. Iron, arsenic, and quinine, especially in the cases complicated with malaria, pro-

duced a distinct but temporary improvement. Free opening of the bowels with magnesium sulphate or castor oil to counteract the persistent constipation gave considerable relief. The thermocautery and blistering the head and spine with iodine always caused a distinct temporary alleviation of the symptoms.* Otherwise drugs did not appear to do any good. With care, cleanliness, good feeding and proper attention, life may be prolonged for a considerable period, but the end comes sooner or later.

Clinical Records of a Series of Cases of Sleeping Sickness.

Case 1.—Gerako. F. 20. Ad. July 15, 1902. Died July 31, 1902. Native of Buse, Uganda :—

Family History.—Impossible to get. One brother said to come to visit her, apparently healthy. No other cases of Sleeping Sickness said to be in family. Other points not obtainable.

State on Admission.—Found lying on ground outside a native hut in a state of semi-coma; marked tremor of tongue and other muscles. On admission to hospital, lies in bed, takes no interest in anything. Lies more in a state of semi-coma than sleep. Eats a little when forced. Speech practically nil, apparently does not understand. Passes motions in bed.

Circ. System.—P. 120, reg. equal. Vol. small, tens. low. V. W. unthickened. H., u. b., 3rd rib, l. b. 3½ inches from m. s. l., r. b. ¼ inch from m. s. l., A.B. 5th sp., 3 inches from m. l., no visible puls. Aus. Sounds feeble, no murmurs.

Resp. Syst.—R. 22, reg. shallow, thor. abdominal.

Palp., V.F., not impaired. Per note res. both sides all over. Aus. vesic, no prol. expir.

Aliment. Syst.—Bowels constip. Palp. nil, muscles somewhat rigid. Perc. Liver, u. b., r. d., 5th rib, a. d., 6th rib, l. b. c., mar. spleen, u. b., 8th r., l. b. c. m.

Stomach not enlarged.

Nerv. Syst.—Semi-unconscious, not drowsy or sleeping. Tremor very marked in tongue, none in face, seen in muscles of hands and arms; both sides very marked, also legs on both sides; none abdom. or thorax muscles; now and then a coarse twitching in muscles of forearms both sides.

Motor functions. Reflexes. Sup. present, marked plantar, and epigastric deep, knee present, diminished, more marked left than right. No ankle clonus.

Sensory Functions.—Pain, touch, and temp. sense present, not increased, ordinary. Motions passed in bed. Legs weak, not wasted in appearance, no def. paralysis, grip feeble, loss of motor power.

Special Senses.—Pupils equal, react. to light and during accommodation. Smell and hearing unobtainable. No paralysis eye muscles.

Urinary System.—Examination. No albumin, blood or sugar.

Lymphatic System.—Slight glandular enlargement under jaw on both sides, small, also slight in groins, more marked left than right; other situations, none palpable.

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* The injection of a solution of sublimate into the veins, as recommended by Baccelli for other diseases, caused a fall in the temperature, and this condition persisted for several days.

Further Notes—July 26, 1902. Not much change, tremors a little more marked. Does not sleep much, but is very dull and apathetic, mumbles only in reply to questions.

July 30. Worse. Feeds very badly, and, on swallowing milk, has some difficulty; pain in neck on pressure, and apparently pain on swallowing. Marked swelling has now appeared in sub-mental region of right side, tense, and exceedingly tender; extends over mesial line to left. Tremors about the same in extent, no def. signs of paralysis. Motions passed in bed. Temp. not up. Attempt to examine throat impossible, resists strongly, and has apparent difficulty in opening mouth. No physical signs in lungs.

July 31. Died suddenly about 4 A.M.

P.M. 8.30 A.M.; 4½ *Hours after Death.*

External Appearances.—Skin soft to touch. No scratch marks or slightest signs of eruptions. Body well nourished, no emaciation, no wounds or old scars, no bone thickening, large swelling, elastic in consistence, on left side under jaw, extending up into parotid region and slightly over mesial line; glands palpable on both sides of neck, also in rg. axilla, slight in left, also in groin.

Section.—Well marked layers of fat sub-cutaneously over thorax and abdomen.

Thorax.—Retrosternal glands enlarged.

Pericardium.—Small amount of clear fluid in sac.

Heart.—Not enlarged. Valves competent. No alterations in muscles. No atheroma aorta.

Lungs.—No pleurisy or pleuritic fluid. L. L. A little cong. base. No pneumonia. R. L. No pneumonia. Congestion lower lobe.

Abdomen.—No peritoneal fluid. *Liver* slightly enlarged. Normal in consistence. *Gall Bladder.* A little greenish bile.

Spleen.—Enlarged slightly. Slate coloured capsule. Section dark, pigmented.

Kidneys.—Capsule strips easily in both. Section nil.

Pancreas.—Nil.

Intestines.—Small, nil; large, full of fæces. Appendix normal. Ascaris lumbricoides many. Ankylostomes, many in duodenum. Trichocephalus dispar in colon. All mesenteric glands greatly enlarged, matted together in places, some as big as walnuts, hard in consistence, no pus.

Mature F. perstans.—One entire and one portion of an adult in tissue of mesentery near root.

Bladder.—Empty, no urine.

Uterus and Vagina.—Nil.

Ovaries.—Both enlarged. L. three times normal size, R. about twice.

Neck.—Large swelling noted in life, due to a mass of glands under jaw of all sizes. Sub-mental necrotic breaking down. Soft in consistence. All glands in both triangles enlarged. A large gland, size of a big walnut, in post triangle of rg. side, pus in its interior.

Tongue healthy. Tonsils enlarged, follicular tonsillitis. *Larynx*, extreme œdema glotidis, tissues also around œdematous. Thyroid apparently healthy. *Pharynx*, pharyngitis.

Brain.—Dura adherent at places, a considerable amount of clear fluid. Vessels over surface very congested. Pia arachnoid markedly adherent. A large amount of distinctly turbid fluid, especially over the sulci and round the vessels under the membrane. Picture of meningitis not so marked on base of brain.

Section.—Tissue of brain firm. Both lateral ventricles distended and full of fluid, nuclei and thalamus nil. Pons and medulla and cerebellum nil macroscopically. Cord not examined.

Lymphatic System.—General enlargement of glands all over body. Special regions already noted.

Histological Examination.—Old malarial pigment in spleen and liver. No malarial parasites in spleen. Smears (measured) of blood from different organs and vessels. No def. results. Smears from glands, no Filariæ, pus and all glands full of diplococci. Sub-dural and sub-arachnoid fluid, no Filariæ. Pus cells and diplococci.

Brain.—Picture of a meningo-encephalitis. Pia arachnoid, well marked inflammation, considerable leucocytic exudation. Vessels in interior, with their peri-vascular lymphatics, packed with mononuclear leucocytes. Diplococci numerous, but apparently Sleeping Sickness changes first, then complication of micrococcic infection.

Pituitary Body.—Section thro. ant. portion, capsule thickened, some exudation. Interior nil.

Thyroid.—No decrease of colloid material.

Ovary.—Fibroid tissue increased. Hæmorrhagic areas in places.

Tonsil.—Erosion of epithel, covering at places, round-celled infiltration beneath this vessel congested. Increase of monuclear leucocytes.

Glands.—Bronchial, two. One shows very little, also other.

Neck.—One shows intense congestion of blood-vessels, and dilation of those lymphoid tissue increased.

No. 2. More marked than first. Enormous dilation of capillaries engorged with blood.

No. 3. Not much change. No dilated capillaries, increase of lymphoid tissue.

Case 2.—Caperi. M. 8. Ad. July 15, 1902. Dis. Native of Entebbe, Uganda :—

Family History.—Impossible to find out. F. 1 said to have S. S., doubtful. Duration of illness said to be two months.

Pers. History.—Found at Busi, when seen very apathetic and drowsy, nodding his head when sitting; walking impaired, pulls feet along after him resembling locomotor gait. Taken to Entebbe.

State on Admission.—No emaciation, eats, understands when spoken to and obeys directions. Drowsiness marked, but does not really sleep. No tremors visible, glands enlarged. F. P. in blood.

Circ. Syst.—P. 80, reg. equal. Vol. very small, tens. low. V. W. nil, no visible puls. a. b. 5th space u. b. 3rd rib, l. b. mam. line, r. b. ¼-inch, rg. sternal border, aus. sounds faint, no murmurs.

Resp. System.—R. 18 equal, thor. abdomen, no cough. Note res. all over. Sounds vesic. somewhat faint.

Alim. System.—Insp. nil, pal. sp. and liv. palp. Liver u. b., 5th r. a. d., 6th l. b. 1 inch below c. m. Spleen u. b., 7th rib l. b. 1 inch below c. m. Bowels constip.

Nerv. Syst.—Drowsiness, no tremor in tongue or arms. *Mot. F. K.* reflexes pres. ordinary plantar cremasteric, epigastric well marked. No wasting or paralysis of muscles. Walks peculiarly, dragging one leg after other. Muscular power? Ankle clonus nil.

Sens.—Touch pain and temp. sens. present, legs, arms, abdomen.

Spec. Sens.—Eyes, pupils equal mod. dil., react to light and accom., no nystagmus, no ocular paralysis. Hearing apparently unimpaired.

Urinary System.—No subject. symptoms. Quant.? sp. gr. 1020, sl. alkaline. No alb., bl. or sugar. Deposit nil.

Lymphatic Syst.—Glands palpable, ant. post. neck both sides and all other situations, e. g. gen. enlargement.

Further Notes.—July 26. Temp. fluctuating, irreg., pyrexia; condition generally unchanged, retains command over sphincters, passing motions ordinarily.

July 30. Pyrexia to-night, nothing else, eats well and is less drowsy.

August 1. Seems much better, up during day. Walks better and does not drag his limbs so much. Eats well. No paralysis of sphincters. Tendency to drowsiness. No tremors.

August 2. No change except the detection of a very fine tremor now and then on dorsum of tongue.

August 5. Very fine tremor in tongue at times. Knee reflexes present. Now diminished distinctly. Marked tendency to drowsiness to-day. Pupils equal, mod. dilated. Glands, no increase in size since July 15. Sp. punc. 5 P.M.

August 13. No further change. Quinine 5 grs., mag. sulph. ʒ j. twice daily.

August 19. Better, runs about out doors all day, no marked tendency to sleep.

August 28. Great rise in temp. (102). Drowsiness much more marked. General conditions worse.

August 29 and 30. No change.

September 1. Better, not so drowsy.

September 3. No change.

September 8. No change.

September 11. Rather brighter, no tremor tongue or hands, still drowsy look.

September 24. *In statu quo.* Runs about all day. Still drowsy.

September 29. *In statu quo.*

October 5. *In statu quo.*

October 10. No change. Brighter. Stays about doors during day. Not much drowsiness.

October 28. Has remained in same condition since 10th. Still sometimes a heavy dull look, not markedly stupid, however.

Table of Blood Counts.

1902.		
July	24.	R., 3,900,000; L., 6200.
August	6.	R., 3,560,000; W., 8400; Hb., 78 per cent.; P. M. N., 39 per cent.; L. M., 30 per cent.; l., 16 per cent.; E., 15 per cent.; T., 0 per cent.
August	14.	R., 3,500,000; W., 9000; Hb., 81 per cent.
August	19.	R., 3,580,000; W., 7000; Hb., 78 per cent.
September	10.	R., 3,780,000; W., 8700; Hb., 76 per cent.
September	17.	R., 4,440,000; W., 6560; Hb., 80 per cent.; P. M. N., 32 per cent.; L. M., 6 per cent.; l., 39 per cent.; E., 20 per cent.; T., 3 per cent.
October	28.	R., 4,400,000; W., 5000; Hb., 80 per cent.

Case 3.—Waniha. F. 25. Ad. July 15, 1902. Dis. August 28, 1902. Died. *P.M.* Native of Manyamgo, Uganda:—

Family History.—F.? M.? S. l. h. B. O. Ch. l. h.

State on Ad.—Duration of illness not known. Carried in from out district. Very feeble and weak, walking with a very marked shuffling gait. Tremor of tongue marked, also slight in hands. Glands in neck, etc. Pyrexia. No marked sleepiness, but somewhat drowsy and very apathetic. Ad. as an early case, not much wasting.

Circ. Syst.—P. 120. Reg. equal. Vol. small. Tens. practically nil, V.W. unthickened. A. B. not. palp. V. B. 3rd rib, l. b. 3½ inches from ms. l. r. b. ¼-inch to rg. of m. s. l. Ausc. sounds faint at apex, better heard at base. No murmurs.

Resp. Syst.—R. 24. Reg. equal, thorax, abdomen. Condition of chest healthy, no dullness anywhere, breath sounds vesicular.

Aliment. Syst.—Appetite good, eats native food. Mouth, nil. Bowels generally constipated. Liver u. b. R. d., 6th a. d., 7th l. c. c. m. Spleen u. b., 7th l. b. cont. margin. Nothing else in abdomen.

Nervous System.—Intellectual functions: Apathetic, not evincing interest in anything except food. Does not sleep much, though at times apparently drowsy. No delirium or coma. Speech slow and unintelligent.

Cranial Nerves.—Smell impossible to obtain. Sight apparently unimpaired; recognises people. No paralysis eye muscles. Nystagmus nil. Pupils equal, mod. dilated. Reacts to light and accommodation. Fifth nerve unimpaired, no paralysis of face muscles, e. g. facial, other nerves not paralysed. No deviation of tongue.

Motor Functions.—No def. evidence of paralysis. Grip feeble and musc. power of limbs also feeble. Co-ordination impaired. Romberg's symptom very slight. Nutrition of muscles somewhat wasted and flabby. Fine tremor on tongue and in upper limbs.

Sensory Functions.—Ordinary senses well marked. Pain sense increased somewhat, temperature sense undiminished. Musc. sense unobtainable.

Reflex Functions.—Plantar abdom. Conjunctival present well marked. Knee reflexes increased a little on left side, about ordinary right, no ankle clonus or knee clonus. Organic reflexes not impaired.

Gait.—Peculiar, slow and shuffling.

Urinary System.—No subj. symptoms, sp. gr. 1029, no albumin Bl. or sugar. Dep. mucus. Quant?

Lymphatic System.—Glands palpable in neck, groins slightly, and in axillæ.

Skin.—No eruptions or any def. pruritus.

Further Notes.—July 22. Very bad. P. 130, almost imperceptible, very weak.

July 25. Somewhat better. Does not pass motions in bed.

August 1. Appears better. Gets up to verandah for food. Walk peculiar, does not raise feet far from ground but pushes them more, tends to stagger a little and has to support herself to get into bed.

August 2. No change.

August 5. Reflexes. Slight increase left as compared with rg., which is about normal. Very apathetic and stupid, not drowsy and no tendency to sleep.

August 13. Quinine 5 grs. Mag. sulph. ʒ ij. twice daily. No further change.

August 14. Diarrhœa since yesterday. Medicine stopped. Emaciation is now becoming marked. Very stupid, heavy expression of face. Rigidity of neck muscles appeared. Painful when touched, tremors increasing. Has remained in bed for four days, *e.g.*, since morning of 10th.

August 16. Emaciating rapidly. Passing motions in bed. Rigidity of neck, commencing contractions of legs. Pulse very feeble, no tension. Very ill.

August 18. Slightly better. Emaciation extreme. Slight pruritus to-day. No eruptions, tremors about the same. Still passes motions in bed.

August 19. Slightly better, took milk this morning readily. Passing motions in bed.

August 24. Extreme weakness. Very low temp. (96 F.). Steady pulse, tremor decreased, knee jerk cannot be elicited. The patient is constipated.

August 26. Several convulsive attacks. The head is pushed backwards. The muscles of the neck are contracted (during the attack only), twitching of facial muscles, more marked on the right side; the eyeballs look to the right, the pupils do not react to light, stertorous respiration. Attack lasts about 2–3 minutes and is soon followed by another.

August 27. Several convulsive attacks like yesterday.

August 28. In the morning a few convulsive attacks. In the afternoon well-marked Cheyne-Stokes breathing, the pulse cannot be felt any more at the wrist. Tremor very much decreased. The patient dies at 6.30 P.M. without showing any more convulsions.

Table of Blood Counts.

1902.
August 6. R., 3,650,000; W., 7500; Hb., 75 per cent.
August 14. R., 3,500,000; W., 8000; Hb., 78 per cent.

P.M. August 29, 15 hours after death (body well preserved, as the night was very cool).

External Appearances.—Extreme emaciation, a superficial and not very large bed sore on right gluteal region, no skin eruptions. Glands palpable on both sides of neck, axillæ and groin.

Thorax.—Pericardium, small amount of clear fluid. *Heart* not enlarged, flabby, valves comp. aorta normal. *Lungs* quite normal. No pleural effusion.

Abdomen.—Relations of organs natural. No peritoneal effusion. Liver slightly enlarged, early nutmeg. Spleen very little enlarged. Slate col. Caps. Sec. dark pigm. Kidneys nil. Pancreas nil. Bladder normal. Intestines, large intestine full of hard fæces. Parasites, many ankylostomes, very few asc. lumbar, no trichocephalus dispar. Mature *perstans* in mesentery. 4 ♀ portion ♂.

Brain.—Dura mater not adherent to the calvarium. Vessels very congested. Longitudinal sinus full of coagulated blood. Pia-arachnoid over the convexities thickened and opaque, no flattening of convolutions. Cerebro-spinal fluid slightly turbid and in excess. On section tissue of brain firm ventricles not dilated. Pituit. body normal, cerebellum, pons, medulla nothing macrosc. Cord not examined.

Histological Examination.—Brain. Usual mononuclear exudation under pia and round vessels in interior of brain, much slighter, however, than in the other cases. Capillaries congested.

Spleen.—Old malarial pigment.

Liver.—Old malarial pigment, cloudy swelling of cells.

Kidneys.—Cloudy swelling of epithetial cells.

Mesenteric Gland.—Some thickening of capsule, no congestion or dilation of vessels. Lymph. tissue apparently normal.

Case 4.—Tounionza. F., 26. Ad. July 18, 1902. Dis. September 30, 1902. Died. *P.M.* Native of Busi, Uganda :—

Family History.—Ch. 2. 1 died of S. S. in hospital No. 5, others said to be healthy. B. & S. O. Half-brother l. h.

Pers. History.—Brought in from Busi. Nothing else obtainable.

State on Adm.—Apparently ill. Tremor of tongue very marked, also in muscles of neck. Arms, fine tremor, legs also. Covered with scabies from head to foot. Lymph. glands in sub-mental region and under jaws. Also in groin, those on rg. side tender. T. 100. P. 86. Glands not very large. No cough or evidence of other disease.

Circ. System.—P. 86. Reg. equal. Vol. small, tens. low. V. W. not thickened. A. B., 5th space, 3½ inches from m. l. u. b. 3rd rib, r. b. 1¼ inches rg. of m. l., l. b. 4 inches from m. l. Ausc. M. A. First sound nil. Syst. murmurs at A. A. and P. A., second nil.

Resp. System.—18. Reg. equal, shallow. Thorac. abdom. Note res. all over. Br. feeble and faint. Vesic. insp. not prolonged.

Aliment. System.—Inspec. nil. Spleen palpable. *Liver* R. D. 5th rib, A. D. 6th, L. B. ¼ inch below C. M. *Spleen* U. B. 7th, L. B. 2 inches below C. M. Ant. to near mid-line.

Urinary System.—July 24. Menstruating exam. postponed. August 5. Quant.? Dep. Muc., sp. gr. 1025. No alb., blood, or sugar.

Lymphatic System.—Enlarged glands post triangles, nil sub-mental, nil axillæ. Shotty in both groins, more so on right than left.

Nervous System.—Very stupid and unintelligent. Heavy silly expression. No marked sleep. No delirium or coma. Will not answer questions well; speech slow and mumbling.

Cranial Nerves.—Smell and sight impossible to determine. No paralysis eye muscles; no nystagmus. Pupils equal, mod. dil. React to light and accom. No facial paralysis or of other cranial nerves.

Motor Functions.—Motor poor, diminished. Co-ordination impaired. Muscles somewhat flabby. Tremor fine marked in tongue and arms and legs. Weakness of legs, no def. paralysis.

Sensory Functions.—Touch unimpaired. Hyperasthesia all over. Temperature sens. about normal. Resists being handled greatly, and gives short cries of pain. Hyperasthesia of 5th nerve where branches come out.

Reflex Functions.—Plantar, present good, also other superficials. Knee exaggerated both sides, rg. more than left. *Organic.* Passes water in bed, also motions, this however probably due to inability to get out of bed. No ankle clonus. *Gait.* Peculiar, slow, and shuffling. *Throat, nose,* etc., nil.

Further Notes.—August 1. A little worse; very dull and stupid. Resists being touched very much. Evident pain menstruating, and passing water in bed. Drowsiness marked.

August 5. Slightly better, very dull and stupid. Eruptions diminishing greatly under sulphur and cleanliness. Knee reflexes still very exaggerated both sides. Spleen punct. 5 P.M. Yesterday and to-day has ceased passing motions in bed, so probably no paralysis of sphincters, only weakness and no proper nursing facilities.

August 13. Q. grs. 5. Mag. sulph. ʒj, twice daily; eruptions much better. Much better again, gets up daily and eats well.

August 18. No change.

August 25. Much worse. Dull apathetic expression. Patient does not get up. Tremor much increased. Knee reflexes exag., especially right side.

August 26. No change.

August 28. A little better.

August 30. No change.

September 2. Better. She is able to get up. Tremor of tongue and hands well marked. Knee reflexes still exag., espec. right side.

September 8. About the same. Med. stopped.

September 11. Tremor in tongue very marked. Also in arms, emaciation becoming marked. Goes about now outside all day.

September 18. Worse again. Very drowsy. Taken to bed. Passing motions in bed. No eruptions now. Emaciation extreme.

September 23. Looks a little better, brighter, does not leave bed now, however. Tremors very marked. Emaciation extreme. Temp. subnormal. Reflexes still sl. exag. about the same.

September 24. Very apathetic. Tremor in tongue and hands very marked. Looks bad.

September 25. Much the same. Lying in bed. Extremities very cold.

September 27. Semi-conscious. Not sleeping, can be roused by stimulation.

September 28. Semi-conscious. Mucus welling from mouth and nose. Very bad. Reflexes still present, but now greatly diminished. Pulse imperceptible at wrist. Very cold, especially extremities. Resists being touched, which apparently causes great pain. Cries out.

September 29. In a state of coma, almost complete; can be roused only with great difficulty. Temp. very low. Motions passed spontaneously. Emaciation marked.

September 30. 8 A.M., complete coma; respirations deep, laboured. Eyes wide open, conj. glazed. Reflexes there still present. Cadaveric smell. 4 P.M., still living. 6 P.M., died.

Table of Blood Counts.

1902.	
August 15.	R., 2,700,000; W., 4300; Hb., 62 per cent.
August 19.	R., 2,160,000; W., 3750; Hb., 60 per cent.; P. M. N., 52 per cent.; L. M., 30 per cent.; l., 14 per cent.; E., 4 per cent.; T., 0 per cent.
September 18.	R., 3,000,000; W., 4300; Hb., 65 per cent.
September 23.	R., 3,160,000; W., 4370; Hb., 65 per cent.
September 24.	R., 3,000,000; W., 4300; Hb., 64 per cent.
September 25.	R., 2,800,0 0; W., 3100; Hb., 64 per cent.
September 26.	R., 2,400,000; W., 3120; Hb., 60 per cent.
September 27.	R., 2,600,000; W., 3100; Hb., 60 per cent.
September 28.	R., 2,720,000; W., 12,500; Hb., 60 per cent.
September 29.	R., 3,100,000; W., 26,800; Hb., 64 per cent.; P. M. N., 72 per cent.; L. M., 15 per cent.; l., 13 per cent.; E., 0 per cent.; T., 0 per cent.
September 30.	8 A.M., R., 4,280,000; W., 25,600; Hb., 75 per cent.; 4 P.M., R., 4,680,000; W., 15,600; Hb., 80 per cent.

Post mortem, October 1, 8 A.M.

External Appearances.—Extreme emaciation. Many pigmented spots, about the size of a florin, all over body. No signs of the old scabies. No bed sores or eruptions.

Thorax.—No pericardial fluid. Heart small. Muscular substance somewhat flabby. Rg. auricle, a lot of p. m. clot, valves healthy; no endocarditis. Aorta healthy.

Lungs.—No pleural fluid. R. L., healthy. No congestion; some œdema of base of lower lobe; no bronchitis or pneumonia. L. L., a little œdema and congestion of lower lobe.

Abdomen.—No peritoneal fluid. Exam. of fæces just after death showed ova of ascaris, ankylostoma, oxyuris vermicularis, and Bilharzia.

Liver.—A few adhesions to diaphragm. Old malarial pigmentation.

Spleen.—Enlarged markedly. Capsule slate-coloured. Section pigmented, chronic malarial.

Kidneys.—R. Capsule strips easily; substance normal. L. ditto.

Stomach.—Nil. Duodenum a few punctiform points; no ankylostomes there, but many in upper part of jejunum. Intestines nil. Rectum, four small ulcerated hæmorrhagic spots small in size, scraping many Bilharzia ova. Embryo adults not found.

Parasites.—Ascaris lumbricoides; many ankylostomes; many trichocephalus dispar. Many bilharzia. Ova numerous. *Bladder* healthy. *Uterus* healthy. Vagina, some old inflam. and ulcerative change near vulva.

Neck.—Nil.

Lymph. System.—Enlarged in all areas, pea to brazil nut, four posterior abdom. ones as large as walnuts, very hard.

Head.—Calvarium not thickened. *Dura.* Some congested vessels on upper aspect. No excess sub-dural fluid and no adhesions. Pia arachnoid clear; no excess of fluid. Veins over vertex congested. Very little macroscopic change; other parts of brain no change noticeable; cord not taken out. Lat. ventricles not dilated, contained a little fluid.

Histological Examination.—Spleen and liver much malarial pigment. Brain presented same appearance as former cases. Meningeal exudation, and also perivascular infiltration in substance, latter not very marked.

Case 5.—Yaro. M., 35. Ad. July 18, 1902. Dis. September 4, 1902. Died. *P.M.* Native of Buse, Uganda:—

Fam. Hist.—F., d.? M., d.? B., l., 40. S. 1, l. h., ch. 0.

Pers. Hist.—A labourer, brought in, said to be suffering from Sleeping Sickness.

State on Admiss.—Heavy, stupid, unintelligent man, speech slow and thick. Some emaciation, skin dry and harsh, covered with old scabs and sores, pruritus; scratching himself. Enlarged glands, triangles of neck, sub. max., sub-mental, also groins and axillæ. Tremor fine in tongue, also slight in hands, not in legs. T. 99, P. 88, R. 16. Walk typical slow, and pushes feet along ground rather than raising them.

Circ. Syst.—P. 100, reg. eq., vol. very small, tens. low, V. W. unthickened. *H.*, u. b., 3rd r., r. b., 1 inch to rg., m. l., l. b. 4 inches to lf., m. l., a. b., 5th, s. p. 3½ inches lf. of m. l. *Aus.* Sounds faint, espec. at base, no murmurs.

Resp. Syst.—R. 16, reg. eq., thor. abdom. Insp. nil. Palp. reson. Note all over. Ausc. Vesicular ordinary.

Aliment. Syst.—Tongue small, pointed, lips not thick, fur on dorsum. Appetite good. Bowels constip. Abd. nil on insp. *Liver*, u. b., r. d., 6th r., a. d., 7th r., l. b. c. m. *Spleen*, u. b., 8 r., l. b., 12th r. Ant. mam. line, enlarged.

Urinary Syst.—Sp. gr. 1018, no alb., blood, or sugar. Straw-coloured. Slight deposit of mucus.

Skin.—Rough, dry, harsh, old scratch marks. Covered with old dried-up pustular scabs all over body. Always scratching himself.

Lymphatic Syst.—Glands enlarged generally.

Nerv. System.—Heavy, dull, stupid, slow speech, imperfect articulations. No delirium or marked tendency to sleep, but drowsy and stupid. *Cranial* nerves, no paralysis evident.

Motor Functions.—Muscular nutrition poor, though no marked emaciation. Grip feeble for a man of his age and development. No paralysis. Incoordination impaired distinctly. Romberg's sign present. Other coordinated movements impaired also. Fine tremor tongue and arms, none in legs. No rigidity or contraction.

Reflex Functions.—Knee reflexes present. More marked left than right. No ankle clonus. Superficial present, slight.

Sensory Functions.—Pain, touch, and temp. present; no hyperasthesia.

Special Senses.—Eyes, pupils equal, mod. dil., react. to light and accom. No nystagmus. No ocular paralysis.

Further Notes.—August 14, 1902. About the same, pulse increased in frequency, 120, very small vol. and low tension. Sounds very faint, tremors about the same.

August 25. In the last few days patient has got much worse, much wasted, extreme weakness, bedridden.

August 29. Sub-normal temp, thready pulse, drowsiness very marked; he sleeps also much more than he used, no contractures, no convulsive fits.

August 31. No change.

September 3. Very ill, about dying. Cold, sub-normal temp. comatose. Eyes open, conj. reflex still present, slight. Saliva dribbling from mouth. Unconscious. Emaciation becoming very marked. No fits. Pulse thready, almost imperceptible. Motions passing spontaneously. Jaundice of conj. appeared yesterday.

Table of Blood Counts.

1902.		
September 11.	R., 3,875,000; W., 8,700;	Hb., 79 per cent.
September 18.	R., 4,360,000; W., 10,000;	Hb., 85 per cent.
October 9.	R., 4,200,000; W., 8,000;	Hb., 84 per cent.

September 4. *Died about* 6 A.M. *Post-mortem examination at* 10 A.M.

Ext. Appearances.—Body considerably emaciated. Skin dry and harsh. Pig. scars all over body, especially legs; no bed sores. Jaundice in conjunction.

Thorax.—No pleural effusion. Old adhesions upper part of left lung. *Left* lung healthy. *Right* lung rather deeply congested. *Pericardium*, no fluid. *Heart* substance, degenerated, flabby. Valves competent, aorta healthy.

Abdomen.—Fat on section, very yellow icteric staining.

Spleen.—Enlarged, perisplenitis, old. Capsule slate-coloured. Section, congestion and pigmentation.

Liver.—Enlarged, adherent to diaphragm; old perihepatitis. Section, congestion, and fatty infiltration.

Gall Bladder.—Much distended. Duct blocked by a soft dark coloured gall stone, also several smaller ones. Contents of gall bladder black, grumous liquid. Opening into duodenum patent.

Stomach.—Hour-glass constriction, enlarged. Inner surface no old ulceration. Part opposite constriction thicker than rest. Duodenum and intestines contain much slimy mucus, otherwise normal. Cæcum loaded with hard fæces. *Parasites.* Ankylostomes plenty, also Trichocephalus dispar; no Lumbricoides.

Bladder.—Nothing to note.

Kidneys.—R. capsule strips easily. No inflam. L. ditto.

Pancreas.—No congestion, normal in consistence.

Mesentery.—Numerous enlarged glands, from pea to hazel nut.

Neck.—Tonsils slightly enlarged. Larynx healthy. Glands of a general small size. Sub-mastoid. Sub-maxillary. Sub-mental, and in the triangles. No suppuration or breaking down. Thyroid, nothing to note.

Head.—*Scalp* fat, markedly yellow, bile stained. *Calvarium* normal consistence. *Long Sinus* contains some fluid blood, and also a dark red clot. *Dura Mater.* Slightly adherent in places in middle line, otherwise not. Slight excess of fluid. Pia arachnoid can be stripped easily without erosion. Vessels on vertex much congested. Sub-pial fluid in excess, not turbid, markedly bile-stained. At places recent lymph. No pus or suppuration. Convolutions no flattening and no adhesion of Pia in sulci. Appearance of Pia arachnoid membrane somewhat opaque. A considerable amount of fluid in ventricles. Pons, Pituitary body, Medulla and Cerebellum, nothing of note macroscopically. *Cord.* Dura not adherent. No congestion of vessels. Pia strips readily. Substance of cord apparently healthy macroscopically.

Histological Exam. Brain.—Usual exudation on meningeal surface, slight round vessels in substance.

Pons.—Slight exudation round vessels.

Medulla.—Similar exudation, and about same in amount.

Spinal Cord.—Cervical region. Meningeal exudation, also in some vessels in grey matter, not in white. Other regions similar.

Sciatic Nerve.—Degeneration ?

Cerebellum.—Usual exudation in sulci and on outer surface.

Stomach.—Catarrh of mucous membrane. Wall hypertrophid, especially sub-mucous layer.

Case 6.—Kasokabilia. M. 32. Ad. July 18, 1902. Dis. August 31, 1902. Died. *P.M.* Native of Buse, Uganda :—

Fam. Hist.—F. l. h. M. d. B. 2. l. h. S. O. Ch. 2. d. ?

Pers. Hist.—Labourer. Prev. ill, nil. ex. s. pox.

State on Ad.—Big burly negro, pitted with old small-pox marks. Heavy expression, not drowsy. Very marked tremor in tongue and arms, legs fine in character. Walk impeded, puts feet down all right, rather shuffling. Old scars and dried spots, one large scar on right buttock, one on left knee, many on legs. Old spots limbs and body. Glands small in both triangles, also slight under jaw. Shotty and hard enlarged glands both groins. Skin rather dry and harsh, lustreless. Eats well. T. 102° F. P. 100. R. 20.

Circ. System.—P. 100, Rg. equal, small vol., low tens., V. W. unthickened. a. b. 5th space, 3 inches from m. l. u. b. 3rd rib, r. b. 1 inch rg. m. l. l. b. 3½ inches from m. l. at 4th rib. Sounds faint, no murmurs, better heard base than apex.

Respir. Syst.—R. 20 equal reg., thoracic abdominal. V. F. normal. Note resonant all over breathing, ordinary vesicular, no prolongation expiration.

Aliment. System.—Tongue flabby, dirty fur. Bowels constipated. Abd. nothing on inspection. *Liver*, u. b., r. d., 5th, a. d. 6th l. b., c. m. *Spleen*, u.b. 7th, l. b. 12th ant c. m. in mam. line.

Urinary System.—Sp. gr. 1012, no alb., blood or sugar, deposit nil. No subjective symptoms.

Skin.—Old scars buttocks, limbs, knee, and lower leg. Skin harsh and dry, no pruritus.

Lymph. System.—Small enlarged glands in triangles of neck and sub-max. hard in consistence ; also in both groins and axillæ.

Nervous System.—Not specially dully or drowsy. Answers questions intelligently. *Cranial Nerves.* No paralysis in evidence.

Motor Functions.—Legs, muscles flabby. No marked emaciation of any part of body. No incoordination. No Romberg's symptom. Tremor very fine marked in tongue, hands, arms, and legs. Very noticeable feature of case on inspection.

Sens. Functions.—No impairment, touch, temp. or pain sense. Pain sense increased, some hyperæsthesia in limbs.

Reflexes.—Superficial present. Knee equal. Slightly diminished. No ankle clonus.

Special Senses.—Eye, no ocular paralysis. Pupils equal, mod. dilated, react to light and accommodation. Smell unimpaired, hearing also.

Further Notes. July 25. Tremors very marked, increased, eats well and gets up daily and goes outside.

August 1. Much the same, tremors still very marked, tongue, arms, body and legs.

August 7. Worse. Taken to bed to-day and has not been up. Still eats well, tremors as before.

August 12. Never out of bed since 7th, passed water in bed to-day for first time, due to weakness and inability, probably did not ask for urine dish. Very weak and getting rapidly worse.

August 13. Passed water and fæces in bed to-day. *Circ. Syst.* A. b. not palp. borders as before. Sounds almost indistinguishable. P. 130, pract. no vol. or tension, almost impossible to feel. *Resp. System.* Nothing in chest, reson. note at bases and no prolongation in expiration or auscultation.

Alim. Syst.—As before.

Nerv. Syst.—For last six days tremors have increased and are now so marked as to shake bed clothes. Not coarse, still fine in nature. Some rigidity of neck, and painful when touched. Contracture of legs, flexion of thighs on abd. and legs on thighs appeared six days ago and is now marked, cannot straighten them, and cries out with pain when one tries to do so. Reflexes difficult to obtain owing to contracture, but still present.

Sens. Functions.—Some hyperæsthesia about neck and marked pain in legs. Temp. sense unimpaired.

Spec. Senses.—Eyes wide open. Pupils equal, mod. dil., react to light and accom. Patient very weak and apparently near end, still eats of own accord.

August 14. Still living. Sordes on gums and teeth, foul smell, very weak. Urine drawn off by catheter, a little blood from urethra, which accounts for resulting slight trace of albumin. No sugar.

August 15. Slightly better.

August 16. About same, extremities cold, pulse very feeble, passing motions in bed.

August 18. Still living. Tremors excessive. Eyes wide open, no paralysis definite. Passing motions in bed.

August 19. Pulse imperceptible. Extremities cold. Much purulent discharge welling out from nose, passing motions in bed. Marked leucocytosis suddenly appeared to-day.

August 23. Great fall in temp., extreme weakness. A skin eruption with large blebs on the left forearm, liquid of blebs clear, containing very few leucocytes.

August 24. No change.

August 25. The contents of the blebs has become purulent, dressing with sublimat.

August 27. General conditions much worse. Pulse almost imperceptible, respiration frequent and shallow, less tremor.

August 28. No change.

August 29. No change.

August 30. Pulse imperceptible, respiration shallow and very frequent (44), extremities cold.

August 31. The patient died suddenly about 5 A.M.

Table of Blood Counts.

1902.

August 14. R., 3,812,000; W., 5400; Hb., 75 per cent.

August 15. R., 4,000,000; W., 6870; Hb., 78 per cent.; P. M. N., 56 per cent.; L. M., 28 per cent.; l., 12 per cent.; E., 1 per cent.; T., 3 per cent.

August 19. 4 P.M. R., 5,200,000; W., 33,125; Hb., 90 per cent.; P. M. N., 70 per cent.; L. M., 20 per cent.; l., 8 per cent.; E., 0 per cent.; T., 2 per cent.

August 20. 4 P.M. R., 6,200,000; W., 22,000; Hb., 100 per cent.; P. M. N., 70 per cent.; L. M., 17 per cent.; l., 13 per cent.; E., 0, per cent.; T., 0 per cent.

P.M. 10 A.M., 5 *hours after death.*—Body very emaciated. Bed-sores on trochanters. Glands palpable, neck, axilla, groin.

Thorax.—Relations of organs natural. Pericardium appears normal, a very small amount of clear fluid. *Heart* not enlarged. Flabby, much fat. Valves competent. Nothing to be noted in the aorta. *Lungs* normal, no pleural effusion.

Abdomen.—Relation of organs natural. No peritoneal effusion, mesenteric glands enlarged. *Liver*, not enlarged, normal. *Spleen*, slightly enlarged, capsule slate-coloured on section, very dark pigment. *Kidneys*, nil. *Pancreas*, nil. *Bladder*, nil. *Intestines*, large intestine full of hard fæces. *Parasites*, many ankyl., few asc. lumbr., no tric. dispar.

Brain.—Calvarium dense and hard, symmetrical. Dura mater not adherent, vessels very congested. Longitudinal sinus full of coagula. Pia arachnoid over the convexities opaque, can be stripped without erosions, no flattening or wasting of convolutions, cerebro-spinal liquid a little turbid and in excess. On section tissue of brain from ventricles not dilated. Pit. body normal. Cerebellum, pons, medulla appear normal. Cord looks healthy macroscopically.

Histological Examination.—Brain. Picture of usual Sleeping Sickness, meningo encephalitis. Infiltration of small cells under pia, mononuclear lymphocytes spreading down the sulci. Vessels in substance of brain packed round with a layer of mononuclear leucocytes. No other changes visible. Similar changes in pons, medulla, and cord.

Case 7.—Msavika (No. 1). F. 12. Ad. July 31, 1902. Dis. September 16, 1902. Died. *P.M.* Native of Buse, Uganda :—

Faml. Hist.—Unobtainable.

State on Adm.—Peculiar-looking child. Standing, fine tremor seen. Left forearm flexed at elbow and held with extensor surf. uppermost, drop wrist. Other arm normal as to position, fine tremor in both. Walks peculiarly, dragging limbs, also walks with toes of left foot in air. This foot badly infected with jiggers, probably explains position. Tongue, fine tremor. History of headache, but none now. Enlarged glands in post. triangle of neck and under jaws, also inguinal glands shotty in consistence. Skin smooth, no pruritus or eruption of any kind. Passes motions normally. Some rigidity of neck, especially on left side.

Circ. System.—P. 98. Reg. equal. Vol. small, tens. low, V. W. nil. A. B. 6th sp. 3½ inches from m. l., u. b. 3rd rib, l. b. 4 inches from m. l., r. b. 1 inch to rg. m. l. Ausc. Sounds distinct, no murmurs.

Resp. System.—R. 16. Reg. equal. *Aus.* Vesicular, note reson. all over.

Alim. System.—Insp. nil. *Liver* u. b., r. d. 5th, a. d. 6th, l. b., c. m. *Spleen* u. b. 7th, l. b. 12th r. *Bowels* constip., motions passed normally.

Urinary Syst.—Quant.? Dep. muc., sp. gr. 1024. No alb., blood, or sugar.

Nervous Syst.—Rather frightened-looking child. No drowsiness or marked sleep tendency. No coma or delirium. Speech unaffected. *Cranial nerves*, no paralysis evident. *Motor Functions*, marked wasting and feebleness of leg muscles and arm. Grip feeble. Fine tremor tongue, arms and legs. Left arm held in peculiar position, flexed at elbow and wrist hangs. Can be straightened, but some difficulty (contracture). No incoordination.

Sensory Funct.—Unimpaired generally. Some hyperæsthesia locally in legs and arms. No pains in head. Pain complained of in rg. side over kidney.

Reflexes.—Superficial slightly diminished, knee present ordinary, rg. increased left. No ankle clonus. Organic nil.

Special Senses.—Eye, no ocular paralysis. Pupils equal contract to light and accommodation. No nystagmus.

Skin.—No eruptions or pruritus, skin smooth and glossy.

Lymphatic Syst.—Enlarged glands, neck shotty in ant. and post. triangles. Submental, groins also.

Further Notes.—August 2. No change.

August 5. Pain over rg. side gone. No headache. Tremors dist. less. Left knee reflex distinctly exagg., rg. very slightly increased. No ankle clonus.

August 13. Quin. 5 grs. Mag. sulph. ʒ j. twice daily.

August 14. Not so well, tremors increased again. Left arm more contracture. Tendency to contracture in limbs.

August 25. Much worse. Bedridden. No drowsiness.

August 30. Patient appears in great pain, crying out frequently, half unconscious. Does not answer to questions.

August 31. A little better, does not cry any more.

September 2. Better.

September 3. No change.

September 5. Much worse. Every now and again gives sharp cries. Tremors much more. Does not leave bed. Feeds with difficulty. Passing motions in bed.

September 8. Worse. Lies in bed. Groaning and crying out. Tremors very marked. Emaciating. Eyes wide open. Passing motions spontaneously.

September 11. Very bad. Lying in a semi-comatose condition, with eyes wide open. Contracture of legs excessive, calf on thighs and latter on abdomen. Contracture in left arm more marked than when came in. Emaciating rapidly. Large deep bed sore appeared on left trochanter with slough in centre. Does not answer questions or appear to hear when spoken to. Has stopped crying out. Motions passed in bed. Nothing cardiac or respiratory.

September 15. Very ill. Mucus welling from mouth. Semi-comatose, does not understand. 6 P.M. comatose, cold extremities, sub-normal temp.

September 16. Died 3 P.M. No fits, continued in state of coma.

Table of Blood Counts, &c.

1902.		
August	20.	R., 3,080,000; W., 4900; Hb., 66 per cent.; P. M. N., 54 per cent.; L. M., 14 per cent.; l. 26 per cent.; E., 3 per cent.; T., 3 per cent.
September	12.	R., 2,330,000; W., 5000; Hb., 56 per cent.; P. M. N., 61 per cent.; L. M., 18 per cent.; l., 15 per cent.; E., 4 per cent.; T., 2 per cent.
September	15.	R., 1,630,000; W., 13,120; Hb., 50 per cent.

Post Mortem.—Died 3 P.M. *P.M.*, 4 P.M., September 16.

Ext. Appearances.—Much emaciated. Large bed sore over left trochanter, Another one over upper part left buttock. Several smaller ones over dorsal region of back, left side. Skin dry and scaly, no old scars, no scratch marks, no eruptions.

Chest.—Pericardium, no fluid. Valves of heart competent. Musc. substance not degenerated. Pale in colour. *R. Lung* congestion and some consolidation of lower lobe early. Still floats in water. Substance of rest of lung, very little blood, some œdema. *L. L.* no congestion, bloodless, some œdematous fluid. Pleural cavity no fluid, many old adhesions.

Abdomen.—Old adhesions round spleen and liver. Omentum adherent to liver. No fluid. *Liver*, ordinary size. Tissue normal in appearance. *Gall Bladder* contained a little bile, green colour. *Spleen* enlarged slightly, pigmented substance and capsule. Pancreas and kidneys normal. *Stomach* normal. Intestines full of hard fæces. *Parasites*, no ankylostomes. Many trichocephalus and ascaris umbricoides. Uterus and bladder nil. All organs very anæmic.

Neck.—Thyroid and larynx nothing, enlarged gls. hard in consistence in all neck areas, groins, femoral and other regions.

Brain.—Calvarium rather thin. Dura not adherent, no fluid. Sub-dural vessels congested. Sinus, some dark black blood. Pia-arachnoid not opaque, strips easily, practically no excess of fluid. Substance of brain and other parts show practically nothing macroscopically. Cord not examined. No excess fluid in ventricles.

Histological Examination.—Spleen, old malarial pigment. Pig. leucocytes and a few malig. rings. *Lung*, pneumonia rg. lower lobe. *Brain*, lymphocytic exudation very marked round vessels of brain, motor area. Medulla, very great infiltration round vessels, also exudation on meningeal surface. *Pons.* similar. Case presents features of an excessive infiltration round vessels of central nervous system.

Case 8.—Msavika. F. 35. Ad. July 31, 1902. Dis. September 24, 1902. Died. *P.M.* Native of Busi, Uganda :—

Family History.—F. ? S. 1. L. Ch. 0.

State on Ad.—Stupid expression, dull and apathetic, fine tremor in tongue, none in arms. Walk slow and unsteady, tendency to cross feet. Glands enlarged post triangles neck, also both groins, size of beans, none sub-mental. Harshness of skin on arms, on body soft. No pruritus or eruptions.

Circ. System.—P. 90. Reg. equal, vol. small, tens. low, V.W. unthickened. a.b. not pal., u.b. 2nd rib lower bord. R.B. 1 inch to rg. m.l.l.b. 5 inches to l. of m.l. Sounds at apex very faint, no murmur. More audible, but still feeble at base, no murmurs.

Resp. Syst.—R. 18. Reg. eq. Thor. abdom. No cough. Note reson. Ausc. nil.

Alim. System. Liver u.b. R.d 5th, a.d., 6th l.b., c.m. *Spleen*, palp. u.b. 7th, l.b. just below c.m. *Bowels* constip.

Nerv. System.—Excessively stupid. Lethargic and apathetic, no marked tendency to sleep.

Cranial Nerves.—No paralysis.

Motor Funcs.—Slight wasting legs, consistence flabby. Fine tremor, tongue, hands, arms and legs. No incoordination. No paralysis, walk heavy, but does not stagger.

Sens. Functs.—Unimpaired, touch pain and temp. sense not diminished or exaggerated. *Reflexes*, superficial present well marked. Knee reflexes both increased, equal slight. Organic not impaired. Ankle clonus nil.

Special Senses.—Eye. Pupils equal, mod. dilated, react to light and accommodation. No nystagmus. No ocular paralysis.

Skin.—Harsh and dry, no papular eruptions.

Lymph. Syst.—Glands enl. both triangles neck, groins, sub-maxillary, sub-mental, and axillary.

Urinary Syst.—Sp. gr. 1015. No alb., bl., or sugar.

Further Notes.—August 5. Stupidity increased. Doses a good lot, obeys when told to do anything, very slowly. Appetite very good.

August 13. Q. grs. 5. Mag. sulph, ʒ ij., twice daily.

August 25. Very drowsy appearance. Patient has lost flesh in last days. Knee jerk increased, especially on left side.

September 3. No change.

September 5. Emaciation becoming very marked. Tremors increased. Skin very dry, harsh, pruritus, though no eruptions. Much feebler, walk tottering and difficult.

September 17. Took to bed yesterday, has become bedridden. Very drowsy and stupid, emaciation proceeding rapidly.

September 23. Completely bedridden. Passing motions in bed. Emaciation extreme. Knee reflexes slightly increased, left more than right. L.P. done.

September 24. Very ill. Pulse imperceptible at wrist. R. 36, deep and laboured. Conj. glazed. No pneumonic signs.

10 A.M. Skin extraordinarily rough and thick. Very cold. Temp. sub-normal.

3 P.M. Died suddenly. No fits or convulsions.

Table of Blood Counts.

September 17. R., 2,620,000; W., 7800; Hb., 62 per cent.
September 23. R., 1,950,000; W., 13,000; Hb., 42 per cent.

Post-Mortem Examination. Died 3 P.M. *P. Mortem* 4 P.M.

Ext. Appearances.—Body cold; very much emaciated. No old scars or bed sores. Skin very rough and harsh all over body. Some old scratch marks. No eruptions of any sort.

Thorax.—Pericardium, no fluid. *Heart.* Some p. m. clot in rg. ventricle. Muscular substance pale, firm in consistence, no degeneration. Valves competent. No atheroma or disease of those or aorta. *Lungs.* No pleural fluid, a few adhesions, old, on rg. side. *Lung, R.* Pale and bloodless. No congestion or pneumonia. A few collapsed portions near pleura. *Lung, L.* Ditto to rg.

Abdomen.—No peritonitis. *Liver.* Not enlarged, section normal. *Spleen.* Slightly enlarged, section dark red, capsule not markedly slate-coloured.

Stomach.—Some thickening of middle layer at pyloric orifice. Mucous membrane healthy.

Pancreas.—Nil.

Kidney, R.—Very small. Capsule strips with some difficulty. Substance pale, no inflam.

Kidney, L.—Ditto to R., and of same size.

Uterus and Ovaries.—Nil.

Bladder.—Distended, mucous memb. ordinary.

Intestines.—Contained somewhat liquid dark coloured fæces. Appendix normal. Parasites, plenty Ankylostomes, no trichocephalus or ascaris lumbricoides. Mesenteric glands, small enlarged. No mature filariæ found in mesent.

Neck.—Slightly enlarged left tonsil. Tongue, larynx and pharynx all healthy.

Lymphatics.—General enlargement. Glands discrete. Small in size, average a small bean, hard in consistence, no suppuration anywhere.

Head.—Calvarium unthickened. Sub-dural fluid much in excess. L.S. some dark blood clot. Veins over vertex full of dark blood, congested. Some capillary congestion also. Pia arachnoid not opaque, clear, but markedly adherent, especially in sulci. Some excess of its fluid at places. Causes a little erosion at places when stripped. Excess of fluid in ventricles, and both lateral ones slightly dilated. Pituitary body, pons, medulla and cerebellum nil macroscopically. Spinal fluid drawn off by lumbar puncture in excess. Cord not removed.

Histological Examination. Spleen.—Very old malarial pigment, no parasites.

Brain.—Presents ordinary changes, not excessive Mononuclear leucocyte exudation round vessels and on meningeal surface of brain. Other organs, some leucocytic infiltration in substance of heart. Liver cloudy, swelling.

CASE 9.—Kagawa. F., 18. Ad. August 11, 1902. Native of Busoga:—

Fam. Hist.—F. d. k., M. d. fever, B. m., 1. d., sl. sickness. Ch. 0.

Pers. Hist.—Six months ill. Began then to be drowsy, had to be awakened in mornings. No illness before.

State on Adm.—Good-looking woman, fairly intelligent, slight tremor of tongue, none in arms. Not drowsy. T. 103, P. 130, R. 28. No emaciation. Glands small in neck, all areas, specially on left, also sub-maxillary region. Groins, big glands both sides, some as large as small walnut. Slight drowsy and heavy expression. No rash or pruritus.

Circ. Syst.—P. 130, reg. equal, vol. small, tension low, V. W., unthickened; a. b. palpable, 5th sp., 3½ inches from m. l., u. b. 3rd rib, l. b. 4 inches from m. l., r. b. 1 inch rg. m. l. Ausc. Sounds distinct, no murmurs.

Respir. System.—28, reg. equal, sl. hoarseness. V. F. not increased. Note resonant. Sounds normal.

Alim. System.—Tongue dirty, heavy white fur. Appetite good. Bowels constipated. Nothing on inspec. Stomach not enlarged. *Liver* u. b., r. d. 6th, a. d. 7th, l. b. c. m. *Spleen* u. b., 9th r., l. b. 11th.

Urinary System.—No subjective symptoms. 1018. No alb., blood, or sugar. Deposit of phosphates.

Skin.—No pruritus or eruptions. Skin soft in consistence.

Lymphatic Syst.—Large groin glands, hard small glands in all triangles of neck, also sub-max. and sub-mental.

Nervous Syst.—Slight drowsiness. Cranial nerves. No paralysis. Trigeminal points not painful.

Motor Functions.—No wasting of muscles. Leg muscles flabby. Fine tremor only in tongue. No paralysis. Incoordination nil. Sometimes fine tremor in hands. Walk, no marked peculiarities.

Sens. Functions.—Pain sense unimpaired, touch and temp. sense not abnormal.

Reflexes.—Superficial present, ordinary. Knee reflexes absent. Ankle clonus nil.

Special Senses.—Eye, no paralysis, pupils equal, mod. dil., react. to light and accom.

Further Notes.—August 12, 1902. Night temp. 104. Q. 5 grs. Mg. Sulph. ʒ ij. twice daily.

August 13. Night temp. 103·8.

August 14. Temp. keeping up, complaining of splenic pain, and also pain in stomach. Mixture stopped to night, *e.g.*, only one dose to-day. Sp. not palp., U. B. 8th r., not tender; l. b. 12th rib, *e.g.*, some slight increase in size since 11th.

August 19. Complaining of pain in chest and over spleen. Bl. exam., no parasites (malarial).

August 25. Temperature very high, intermittent. Patient complaining of muscular pains in the legs. Heart, base diam. enlarged. Loud, rather harsh systolic murmur, with point of maximum intensity at the apex. Sounds accentuated in the pulmonary area.

August 28. No change in general conditions. The murmur is less harsh.

September 2. T. decreased, much better.

September 8. Distinctly changing for the worse. Tremor now in hands. Losing flesh.

September 23. Much better again. Tremor in tongue very slight. None in hands.

September 29. Much the same. Goes about by day.

October 5. A little worse. Some increase of wasting, not markedly stupid. Goes about by day. Appetite good.

October 10. About the same.

October 28. No change. No emaciation. Looks well. Appears to have gained weight. Still fine tremor in tongue, however; none in hands. Intelligent.

Table of Blood Examinations and Counts.

1902.

August 12. No malarial parasites.

August 19. R., 4,320,000; W., 8400; Hb., 60 per cent.; P. M. N., 62 per cent.; L. M., 14 per cent.; l., 16 per cent.; E., 8 per cent.; T., 0 per cent. No malarial parasites or pigmented leucocytes.

September 18. R., 4,480,000; W., 7000; Hb., 70 per cent.

Case 10.—Basibi. M., 35. Ad. August 13, 1902. Left hospital August 25, 1902. Native of Laji. Sesse group :—

Fam. Hist.—F. d. stomach. M. l. h. B. 2 l. h. 1 died sl. sickness. S. l., swollen arms and legs. Ch. O.

Pers. Hist.—Labourer. No prev. illness.

State on Ad.—Brought in by chief. 20 days ago was noticed to walk peculiarly and would not work as usual, reputed to be drowsy. Heavy, dull, apathetic looking man. Drowsy appearance. Large heavy flabby underlip, tongue very marked tremor. Fine tremor in hands. T., 101. P., 95. R., 20. No emaciation; strong built man; protuberant abdomen; glands nil in triangles, slight under left jaw. Marked in groins, also in femoral group, on left side one soft and rather boggy. Skin harsh and dry. No pruritus or eruptions. Soft sores on foreskin of penis.

Circ. System.—P., 95. Reg. equal. Vol. mod. tens., sl. increase. V. W. thickened.

Heart.—Epigast. pulse. A. b. not palp., u. b. 3rd r. R. B. 1¼ inches r. m. l., l. b. at 4th rib, 4 inches to l. of m. l. Aus. First redup. 2nd ordinary. Slightly faint at base.

Resp. System.—R. 20, equal, reg. V. F. not impaired. Note resonant all over. Sounds. Vesicular all over. No prolongation of expiration.

Aliment. Syst.—Tongue large, soft, and flabby; dirty white fur; lower lip hanging and flabby. Abd. protuberant. Bowels constip. *Liver* u. b. 6th and 7th, l. b. ¼ inch below c. m. *Spleen*, u. b. 7th, l. b. 11th ant. not enlarged.

Urinary System.—Sp. gr. 1026; no alb., blood, or sugar. No deposit.

Skin.—Harsh and dry; a few old spots on back.

Lymphatic Syst.—As in state on admission.

Nervous System.—Distinctly drowsy, stupid heavy appearance. No cranial nerve paralysis.

Motor Functions.—Leg muscles somewhat flabby, not wasted. Incoordination nil. Fine tremor in hands; slight tremor in tongue.

Sens. Functions.—Pain, tem. sense and sensation ordinary.

Reflexes.—Superficial present diminished; knee reflexes absent; no ankle clonus. Walk slow, but not typical.

Special Senses.—Eye, no ocular paralysis. No nystagmus. Pupils equal, somewhat contracted, react slight to accommodation, not to light, very sluggish. Smell unimpaired.

Further Notes.—August 14. Soft sores on penis, no bubo.

August 20. No change.

August 23. Patient has lost flesh; not much drowsiness. Tremor of tongue increased.

August 25. Patient left hospital without permission, and disappeared.

The case is interesting on account of the eye symptoms.

Case 11.—Busoki. M. 40. Ad. August 1, 1902. Dis. September 5, 1902. Died. *P.M.* Native of Buwaya, Uganda:—

Fam. Hist.—F. d. fever, M. d. stomach, B. l. l. h., s. l. l. h. Ch. O.

Pers. Hist.—Labourer.

State on Admission.—Comparatively intelligent looking, not stupid. Not drowsy, no marked tendency to sleep. Not emaciated. Enlarged glands generally small only in neck. Walk typical, does not raise foot, pushes it, shuffling gait. Speech not slow, answers questions quickly. Fine tremor tongue, none in arms. Skin, soft, smooth, no pruritus or eruptions.

Circ. Syst.—P. 90. Reg. eq. Vol. small, tens. slight increase. V.W. slightly thickened.

Heart.—A. B. 5th sp., 3½ inches from m. l., u. b. 3rd r., l. b. 4 inches from m. l., r. b. 1 inch to r. m. l., ausc. redup. first sound, second ordin., no murmers.

Respir. Syst.—R. 16 equal, reg. cost. abdomen, note resonant all over, breathing ordin., vesic., no prolong. expiration No cough.

Alim. Syst.—Tongue, medium size. Some bluish-black pigmented spots on dorsum and frenum. No fur. Abd. nil inspec. *Liver*, u. b. r. d. 5th, a. d. 6th, l. b. c. m. not palp. *Splecn*, u. b. 8th, l. b. 12th, ant. at mam. line, not palpable. Stomach not enlarged. Bowels constipated. Fæces. Many bilharzia ova.

Urinary System.—Sp. gr. 1020. Slight deposit of mucus. No alb., bl., or sugar.

Lymph. Syst.—Chain of small glands in post. triangles, none in groins or submental, some on sub-max. group.

Integ. System.—Some dryness of skin, arms and legs and some pruritus in latter. Skin of trunk, smooth.

Nerv. System.—Not stupid, fairly intelligent. No cranial nerve paralysis.

Motor Functions.—Consistence of muscles diminished. No paralysis or wasting. No incoordination. No R.'s symptom. Grip and muscular power good. Fine tremor, very slight only in tongue.

Reflex Functions.—Superfic., present, slight, knee exagg., rg. ordinary, no clonus, organic normal.

Sens. Functions.—Touch, temp. and pain sense unimpaired, no hyperæsthesia or pain over trigem. points.

Special Senses.—No ocular paralysis. No nystagmus. Pupils eq., somewhat contracted, react perfectly to light and accom.

Further Notes.—August 14. About the same, little change.

August 27. In the last few days an almost sudden change. Extreme weakness, patient is not able to get up from bed, much wasted. Temperature very low, pulse almost imperceptible.

August 29. No change.

September 2. Drowsiness much increased, pulse imperceptible, cold extremities.

September 3. On the point of death, absolutely comatose, only sign of life a slight conj. reflex, temp. sub-normal. Very cold and clammy. No contraction muscles of neck. Saliva dribbling from mouth. Motions passed in bed. No pruritus or eruptions. Skin of trunk still smooth. Reflexes leg gone.

September 4. In a moribund condition.

September 5. Died at 5 A.M. in a state of unconsciousness. No fits, temp. sub-normal.

Table of Blood Counts.

1902.		
August 14.	R., 4,400,000;	W., 5600; Hb., 85 per cent.
September 4.	R., 5,580,000;	W., 9500; Hb., 92 per cent.

P.M. at 10 A.M.

Ext. Appearances—Body not very emaciated. Skin smooth on trunk. Some dryness over arms and legs. No scratch marks or any eruptions or old scars.

Chest.—Pericardium, a small amount of clear straw-coloured fluid. Heart rather small, consistence of muscle good. All valves competent. No endocarditis. Aorta healthy.

Lungs.—No pleural effusion or old adhesions. Some small glands at root about size of pea. *R. Lung.* Generally œdematous. Upper lobe, œdema, congestion and bronchitis. *Middle*, œdema, some congestion, no pneumonia. *Lower* ditto to middle. *L. Lung.* Upper lobe, œdema, congestion, and bronchitis. *Lower lobe*, œdema and congestion.

Abdomen.—*Liver* enlarged, nodular on surface. Some adhesions below it and rg. kidney. *Section* pigmented and full of white fibrous masses, which follow the line of the vessels very markedly. Some of these large areas, some small.

Gall Bladder.—Not distended, contained a little greenish bile.

Spleen.—Enlarged. Old adhesions. Slate-coloured capsule, section darkly pigmented. Much old malarial pigment, no parasites.

Kidneys.—*R.* Capsule strips with difficulty. Cortex not diminished. Substance pale, fatty. *L.* Much larger than right. Capsule strips with difficulty, pale in substance, also fatty.

Pancreas.—Very hard in consistence. A fibroid change round vessels similar to that in liver.

Bladder.—Normal.

Stomach.—Dilated, large, nothing to note in interior.

Duodenum.—Mucous membrane congested.

Jejunum and Ileum.—Congested.

Cæcum.—Congested mucous membrane. Some liquid fæces.

Parasites.—A few ankylostomes, no ascaris or trichocephalus. Glands in mesentery enlarged, size of small peas. No mature F. perstans found. 40 adult Bilharzia in mesentery.

Neck.—Thyroid and larynx normal. En. glands in all neck areas, small in size, hard. Size of peas.

Head.—Calvarium not specially dense. Dura mater, distinctly adherent along middle line. L. sinus, a little coagulum and fluid blood. Pia arachnoid opaque. Sub-pial fluid in excess, clear, pale straw-coloured. No suppuration. Membrane strips without eroding brain surface. Vessels much congested. Section of brain, nothing apparent. Left ventricle much dilated and full of clear straw-coloured fluid. R. lat. ventricle not distended, some fluid. Pituitary body small in size. Nothing macroscopically to be seen in pons medulla and cerebellum. Base of brain presents same picture as vertex. Pia arachnoid opaque with excess of fluid, especially over sulci.

Cord.—Excess of fluid. Congestion of vessels. Dura not adherent: Glands: epitrochlear glands both size of small peas. Groin glands also enlarged about same size.

Histological Exam.—Old malarial pigment liver and spleen, no parasites.

Brain.—Very marked leucocytic exudation mononuclear round vessels in substance, also in the sulci and under pia generally.

Cord.—Similar change to brain quite as marked. Infiltration under pia and very marked round the vessels in grey and white matter of the cord. *Nerve root.*—Medullary substance round axis cylinders of nerves in cauda equina degenerated.

Liver.—Much malarial pigment. Dense masses of old scar tissue. Old gummata.

Pancreas.—Some increase in fibrous tissue at places.

Case 12.—Zenwala. M., 45. Ad. August 18, 1902. Dis. October 18, 1902. Died. *P.M.* Native of Entebbe, Uganda :—

Fam. Hist.—F. d.? M. d.? B. 6, 5 l. h., 1 sus. of early s. s. S. l. l. h. Ch., l. l. h.

Pers. History.—Fisherman. Lives a lot on lake in his avocation. Comes from hill No. 2, where a lot of native huts are. Lives on bananas chiefly.

Pres. Illness.—Headache of late. People who brought him say he has been ill for some time, two years? this undoubtedly incorrect. Within last month has suffered intermittently from headache, pain in chest, legs, and feet. Has been doing no work lately, but just lying about.

State on Ad.—Heavy, stupid looking man, not intelligent, answers questions slowly, speech slow and thick. Heavy upper lip. Walks slowly and unsteadily, shuffling gait. Tongue marked tremor, also hands. Glands, one behind s. mastoid muscle, otherwise not enlarged in neck. Both groin glands large and hard. Skin rough, pruritus.

Circ. System.—P. 80, reg. eq., vol. very small, tens. low, V. W. unthickened. *Heart*, a. b., not palp., u. b., 3rd r., r. b. 1½ inches, r. m. l., l. b. 4 inches to l. m. l. Ausc. Sounds very faint, no murmurs.

Respir. System.—R. 16, reg. eq. Cost. abdomen. Chest, increase in a. p. diameter. Note slightly hyper. resonant in front. Ordinary behind. Breath sounds faint. Vesic., no. ; prolong. expiration.

Aliment. System.—Tongue large, flabby, very dirty, furred. Bowels constipated. *Abd.* Nil, inspect. Liver u. b., 5th and 6th, l. b., 1 inch below c. m. *Spleen*, u. b., 8th, l. b. 12th in mam. line at c. m. Not palp. Stomach not enlarged.

Urinary Syst.—No alb., bl., or sugar. No deposits.

Lymph. Syst.—Glands only one or two in post. triangle, none elsewhere in neck, enlarged in both groins. No venereal signs.

Integ. Syst.—Skin harsh, pruritus. No eruptions or old scars. Old small-pox.

Nerv. System.—Dull, apathetic look. Heavy, unintelligent. Speech slow. Sleep not marked. No cranial nerve paralysis.

Motor Functions.—No emaciation or wasting. Muscular nutrition good. Grip powerful, tremors marked in tongue and hands. Incoordination marked, *e.g.*, R.'s sign, cannot touch point of nose. Tremor increased in hands on putting cup up to lips.

Sens. Functions.—Pain sens. unimpaired, no hyperasthesia. Touch normal, tells accurately where touched. Temp. sense also good.

Reflex Functions.—Superficial present, ordinary. Knee reflexes absent. No clonus. Organic normal.

Eye.—Pupils eq., contracted, accom. to light very badly though slight react. during accommodation, no nystagmus or ocular paralysis. Conj. reflex acute.

Hearing.—Apparently normal, as also smell.

Further Notes.—August 23, 1902. No change.

September 2. Patient has lost flesh, tremor of tongue increased.

September 6. Change for the worse. Tremor increasing. Has come on quite suddenly.

September 8. Very much worse. Tremors very marked now all over body. Can hardly walk. Has to be supported. Emaciating rapidly.

September 17. Bedridden. Took to bed for good yesterday. Tremors increasing. Much worse. Passed motions in bed yesterday.

September 23. Completely bedridden. Tremors as before. Emaciation advancing rapidly. Some hyperæsthesia over neck and on tapping knees. Knee reflexes now present slight. Tremors of legs very marked.

September 24. Much the same.

September 25. A little better, still eats fairly.

September 27. Slight improvement, tremors very marked, however.

September 29. Better again. Crawled out to door yesterday, exceedingly feeble and tottering walk. Tremors excessive whole of body, shakes the bed. Hyperæsthesia when touched on neck, and resists being moved, this apparently causing pain.

October 1. In bed all day, tremors excessive, hyperæsthesia over trigeminal points.

October 5. About the same, has not been out of bed since October 1. Physical signs the same as on first. Purulent discharge from rg. ear. Knee reflexes gone.

October 6. Very ill and feeble, bedridden. Passing motions spontaneously. Still eats.

October 7. 10 A.M. About the same.

October 9. Very ill. Tremors excessive, shaking whole bed. Had a convulsive seizure at 6 P.M. Got rigid, no divergence of eyes, passed off in a few minutes.

October 10. No more seizures. Tremors very marked. Sordes on teeth and gums. Still a discharge from rg. ear. Very ill, still conscious, however.

October 14. Has remained about same. To-night very ill, in a state of semi-coma. L. p. done in evening.

October 16. About the same.

October 17. Very ill. Semi-comatose, breathing laboured.

October 18. Died 4 A.M.

Table of Blood Counts.

1902.

September 17. R., 3,720,000; W., 8400; Hb., 80 per cent. Von Fleischl.
September 25. R., 4,520,000; W., 7800; Hb., 86 per cent.
September 26. R., 4,000,000; W., 6200; Hb., 88 per cent.
September 27. R., 3,800,000; W., 7000; Hb., 84 per cent.
September 30. R., 3,480,000; W., 12,500; Hb., 80 per cent.
October 1. R., 3,200,000; W., 12,000; Hb., 80 per cent.
October 2. R., 3,400,000; W., 10,600; Hb., 80 per cent.
October 3. R., 3,480,000; W., 7500; Hb., 80 per cent.
October 4. R., 3,400,000; W., 8100; Hb., 81 per cent.
October 5. R., 3,100,000; W., 6800; Hb., 82 per cent.
October 6. R., 4,000,000; W., 9300; Hb., 82 per cent.
October 7. R., 4,000,000; W., 6500; Hb., 82 per cent.
October 8. R., 3,600,000; W., 8700; Hb., 80 per cent.
October 9. R., 3,120,000; W., 9300; Hb., 80 per cent.
October 10. R., 3,160,000; W., 11,125; Hb., 80 per cent.
October 11. R., 3,080,000; W., 11,250; Hb., 78 per cent.
October 12. R., 2,900,000; W., 9700; Hb., 76 per cent.
October 13. R., 2,800,000; W., 6500; Hb., 76 per cent.
October 14. R., 3,160,000; W., 8125; Hb., 78 per cent.
October 15. R., 2,800,000; W., 21,800; Hb., 76 per cent.; P. M. N., 72 per cent.; L. M., 10 per cent.; l., 16 per cent.; E., 2 per cent.; T., 0 per cent.
October 17. R., 2,000,000; W., 18,800; Hb., 69 per cent.

Post Mortem. October 18, 10 A.M.

External Appearances.—Emaciated. Superficial bed sore over left trochanter.

Chest.—Heart. No pericarditis or pericardial effusion. Heart, muscle fibre

pale, degenerated. Rg. ventricle full of *post-mortem* clot. Valves all competent. Aorta healthy.

Lungs. No pleural adhesions, no fluid. *R.*, pale in colour, lower lobe slight congestion. *L.*, some slight congestion in lower lobe, otherwise healthy.

Abdomen.—No peritonitis. *Liver* enlarged, fatty and early cirrhosis. *Spleen* enlarged, slate-coloured capsule, old malaria pigment microscopically.

Pancreas.—Nil. *Intestines* not examined for parasites. Fæces showed ankylostoma and bilharzia ova only. *Stomach* slightly distended. *Bladder* normal. *Kidneys* normal.

Lymph. System.—En. glands neck, mesentery, etc., small and hard in consistence. *Neck.*—Nothing abnormal.

Head.—Calvarium unthickened. Dura not adherent. No excess cerebro-spinal fluid. Veins of pia arachnoid slightly congested. Sinus contained some fluid blood. No flattening of convolutions. Pia arachnoid stripped easily, no erosions. No excess subarachnoid fluid. Pia clear in consistence, no opacity. Lateral ventricles not dilated. Macroscopical changes practically then nil. Spinal cord nothing macroscopically. Rg. ear, from which pus came during life, showed no communication with interior of skull, proceeding from middle ear. Other parts of brain nothing macroscopically. Portions kept in Müller, alcohol and formalin.

Histological Examination.—Spleen, old pigment. Fæces, eggs of bilharzia and ankylostoma. Brain: Infiltration of meninges well marked. Vessels in interior surrounded by much cellular infiltration. Special methods: Decrease and atrophy of fibres in motor area. Some loss of Nissl's bodies in the cells. Spinal cord: Marchis stain shows a diffuse degenerative condition of the axis cylinders and sheathes of the nerves not specially limited to any area. Fat deposits in epineurium of the sciatic nerve, and some also in endoneurium.

Case 13.—Sarumi. M., 16. Adm. August 28, 1902. Dis. September 29, 1902. Died. *P.M.* Native of Machogua (near Entebbe) :—

Fam. Hist.—Father and mother both dead, the patient cannot state of what disease. One brother alive and in good health.

Pers. Hist.—No previous illnesses. He was quite well up to two months ago, when he began to complain of headache and drowsiness—onset of illness was gradual—patient states that lately he has become much weaker.

Present State.—September 1. Patient looks drowsy; unwillingness to respond to questions. He answers very slowly when spoken to. Well nourished. A few papules on arms, some pruritus. T. 99. R. 16. P. 110.

Aliment. Syst.—No subject sympt. Tongue furred, fine tremor. Nothing abnormal at the examination of the abdomen. Liver, spleen, not enlarged, no intestinal symptoms. *Micr. exam. of fæces*: many ova of bilharzia.

Circ. System.—No subj. symptoms. Pulse of reg. rhythm, equal, low tension. Apex beat impalp., areas of relative and absolute dulness have normal limits. Ausc. nothing abnormal.

Blood.—Glands enlarged in both sides neck, axilla, groin.

Lymph. System.—

Respir. System.—Resp. cost. abd. regular. Inspec. palp. perc. ausc. of chest, nothing abnormal.

Urinary System.—Exam. of urine. No alb., bl., or sugar. Sp. gr. 1016.

Nervous Syst.—Patient looks drowsy, unwilling to respond to question. No abnormalities of speech. No headache, no giddiness. Cranial nerve functions normal. No paralysis or contractures, no tremor of hands, very slight tremor of tongue. Romberg's symptom absent. Sensory functions normal. Superf. refl. normal. Knee jerk increased both sides. Org. refl. normal.

Special Senses. Eye.—No ocular paralysis, no nyst., pupils eq., ready reaction to light and accom.

Further Notes.—September 8. No change.

September 23. Very drowsy. Fine tremor in tongue, and now in hands. Stays a good deal in bed.

September 27. Drowsiness much more marked, and general state much worse. Tremor increased. Leaves bed very little now. Passed urine in bed for first time yesterday. Pulse just perceptible at wrist. Tens. very low.

September 28. Much worse. Very cold, with sub-normal temperature. P. imperceptible at wrist. Passing motions in bed. Semi-comatose, will not respond to questions. Mucus welling from mouth. 6 P.M. Pulse very slow, 38. In a state of coma; cold extremities. Mucus welling from mouth.

September 29. Died 8.30 A.M. in a state of coma. No fits.

Table of Blood Counts.

1902.	
September 18.	R., 4,200,000; W., 6800; Hb., 80 per cent.
September 28.	R., 4,540,000; W., 8100; Hb., 87 per cent.
September 29.	R., 4,480,000; W., 19,375; Hb., 86 per cent.

Post Mortem Examination. September 29, 9.30 A.M.:—

Ext. Appearances.—Body well nourished, not emaciated. Skin rough and scaly over tibia, otherwise smooth. No old scratch marks, and no eruptions or old scars on trunk. Two old white scars on penis just on skin above glands. Skin around somewhat thickened.

Thorax.—Pericardium, no fluid. Heart, large dilation of rg. ventricle. Left not dilated. Rg. auricle full of *post-mortem* clot extending into rg. ventricle. Valves all competent and healthy. Aorta healthy. No pleural effusion, no old adhesions.

Lungs.—Healthy. *R.* Some œdema of lower lobe, no congestion, bronchitis, or pneumonia. *L.* Nothing abnormal. Bronchial glands enlarged about size of pea.

Abdomen.—No peritonitis or fluid in peritoneum. *Liver* enlarged, pigmented capsule, section pigmentation, and some increase in the fibrous tissue around the lobules. No congestion. *Gall Bladder* contained a little dark-coloured bile. *Spleen* enlarged, pig. capsule, slate-coloured section. Pigmented old malaria.

Kidneys.—*R.* Capsule strips easily. Section normal. *L.* Ditto to. rg.

Pancreas.—Nil.

Stomach.—Slightly dilated, contained some slimy mucus.

Duodenum.—Some congested points, two small mucous polypi.

Jejenum and Ileum.—Lower part some fine congestion.

Cæcum.—Some dark fæces. No ulceration anywhere.

Parasites.—Trichocephalus dispar many, no ankylostomes or ascaris.

Bladder.—Nil.

Neck.—Nothing abnormal. Larynx, vessels and fauces all normal.

Lymph. System.—General enlargement of glands, neck small, size of small almonds. Those in groin and femoral region the same size. Mesenteric slightly larger, and on post. wall of abdomen behind pancreas two very large glands the size of a large walnut. Section hard, no suppuration. Bronchial glands size of peas. Epitrochlear about size of small almonds.

Head.—Calvarium rather thin. Dura mater not adherent to calvarium, slight excess of sub-dural fluid. Veins over vertex extraordinarily congested, turbid, and distended with dark-coloured blood. Smaller capillaries also congested. Pia arachnoid transparent, strips easily without erosion. Sub-pial fluid very slightly

in excess. A fair quantity of fluid in lateral ventricles. Apart from the congestion of the vessels very little macroscopic change. Base similar. Nothing macroscopic in interior pons. cerebellum or medulla. Ventricles not dilated.

Spinal Cord. Dura healthy. Some congestion of vessels underneath. Section nothing apparent macroscopically.

Histological Examination.—Spleen and liver recent malarial pigment. Exam. of fæces. No ova ankylostomes, ascaris. Many ova of Bilharzia. Never blood or ova in urine, so a case of Bilharzial infection of rectal veins.

Case 14.—Semsoni. M. 24. Ad. September 5, 1902. Dis. October 3, 1902. Died. *P.M.* Native of Buse:—

Fam. Hist.—F. d. fever. M. d.? B. 0. S. 0. Ch. 0.

Pers. Hist.—Labourer. Food, bananas, has lived in poor circumstances.

Previous Illnesses.—Has suffered from fever.

State on Admission.—T. 100. P. 96. Heavy dull appearance. Answers questions very slowly. Walk slightly impaired. Very slight tremor hands and tongue. Headache occipital and pain in left side of neck. No pains in chest. Skin slightly rough, no pruritus or eruptions. En. rather large glands in p. triangles, smaller in ant. triangles and submaxillary, also palpable in axillæ, groins and epitrochlear. Not emaciated.

Circ. System.—P. 108, reg. eq., tens. very low. Vol. very small. V. W. unthickened.

Heart.—Epigast. puls. A. B. not palp., u. b. 3rd rib, l. b. 4 inches to l. of m. l., r. b. ¼-inch to rg. of m. l. *Ausc.* Sounds rather indistinct at base. Clear at apex, no murmuring.

Resp. System.—R. 20, cost. abd. Chest well formed. Percus. no dulness. Note reson. all over. *Ausc.* Vesicular ordinary.

Aliment. Syst.—Tongue large flabby. Fur on dorsum. Some small pigmented spots at sides. Fæces nil. Appetite good. Bowels constipated. *Liver* u. b. 5th and 6th, l. b. 1 inch below c. m. *Spleen* u. b. 8th, l. b. 12th, in mam. line at c. m. just palpable. *Stomach* not enlarged.

Urinary System.—Sp. gr. 1018. No alb., bl., or sugar. No deposit.

Lymph. System.—Glands large in size (almond) in post. triangles, also enlarged glands in all the other areas.

Integ. System.—Old scar on rg. side of neck over sterno mastoid. Skin a little rough, no pruritus or eruptions.

Nervous System.—Dull, heavy and very stupid. Speech slow and impaired. Drowsy appearance, but does not actually sleep. Headache in back of head. No cranial nerve paralysis. Tremor tongue and arms.

Motor Functions.—No emaciation or wasting. Musc. nutrition good. Grip fair. Fine tremor in tongue and hands. Incoordination distinct. R.'s symptom. Cannot touch tip of nose accurately.

Sensory Functions.—No impairment.

Reflex Functions.—Superficial present well marked. Knee reflexes brisk, eq. on both sides. No clonus. Organic normal.

Special Senses.—Eye. Conj. anæmic. Trace of jaundice. Pupils small, equal, react poorly to light. Well to accommodation. No nystagmus or ocular paralysis. *Hearing* apparently unimpaired.

Further Notes.—September 10. Worse, drowsiness increasing. Walk distinctly impaired, shuffling in character. Tremors slightly increased.

September 23. Gradually getting worse.

September 24. Tremors increased. Very dull and apathetic. Stays mostly in bed. Walk worse.

September 27. Much worse. Passing water in bed now. Does not get up. Tremor tongue, arms and legs very marked.

September 28. Worse. Emaciation progressing rapidly. Contracture of limbs marked, *e.g.*, legs on thighs, latter on abdomen. Tremors of tongue and limbs worse, knee reflexes gone. Small bed sore on rg. trochanter. Hyperasthesia on being touched on neck or legs, cries out. Rapidly getting worse.

September 29. Worse. Extremities cold. Shrinks greatly when one makes even a pretence of touching him. Does not respond to questions. Not actually sleeping, but approaching coma.

October 1. Very ill, still distinctly conscious. Tremors in tongue not so bad. Contracture of limbs increasing. Very marked hyperasthesia over points of exit of 5th nerve, also shrinks when touched elsewhere. Two small excoriations appeared over front of chest. Passing motions spontaneously. Emaciation marked.

October 2. About the same.

October 3. 11 A.M. Very bad, eyes closed. Breathing deep and laboured. L.P. done 12 noon. Pressure great, fluid gushed out. Breathing became ordinary immediately after. 5 P.M. Breathing deep and laboured again, rapid pulse impercept. Died 6 P.M. in a state of semi-coma.

Table of Blood Counts.

1902	
September 11.	R., 2,771,000; W., 9000; Hb., 65 per cent.; P. M. N., 50 per cent.; L. M., 23 per cent.; l., 20 per cent.; T., 5 per cent.; E., 2 per cent.
September 28.	R., 2,260,000; W., 17,500; Hb., 63 per cent.
September 29.	R., 2,960,000; W., 16,800; Hb., 64 per cent.; P. M. N., 74 per cent.; L. M., 6 per cent.; l., 16 per cent.; E., 2 per cent.; T., 2 per cent.
September 30.	R., 3,300,000; W., 16,800; Hb., 65 per cent.
October 1.	R., 2,500,000; W., 18,750; Hb., 60 per cent.
October 2.	R., 2,600,000; W., 12,600; Hb., 60 per cent.
October 3.	R., 3,400,000; W., 19,300; Hb., 65 per cent.

Post Mortem, October 4. 6 A.M.

Ext. Appearances.—Body much emaciated. Bed sore on rg. trochanter. Two excoriations, one over sternum, and one to the left. Old scar rg. side of neck over sterno mastoid. Skin a little rough, no eruptions or old scratch marks.

Chest.—No pericardial fluid.

Heart.—R. ventricle dilated, left ordinary. Muscular substance pale, slightly degenerated. Valves competent. Aorta healthy. Rg. auricle full of ante mortem clot extending into rg. ventricle.

Lungs.—No fluid in pleura. *R.* Pale, almost white colour. Œdema and some congestion at base of lower lobe. *L.* Pale and bloodless, crepitant, some œdema of lower lobe, no congestion or pneumonia.

Abdomen.—Exam. of fæces, before death, Bilharzia and other ova. No peritoneal fluid or adhesions. *Liver.* Enlarged, pigmented old malaria. *Spleen.* Enlarged, not markedly so. Capsule slate-coloured. Section reddish-brown. Old malaria.

Kidneys.—*R.* Very pale, capsule strips readily. Superficial capillaries infected, as also those in interior. No inflam. changes. *L.* Ditto to right.

Pancreas.—Nil.

Intestines.—Normal. Intestinal parasites. Ankylostoma, plenty, in duodenum

scanty, in upper part of jejunum many, and extending down it for a considerable distance. Ascaris few. Trichocephalus few.

Rectum.—One small patch, blood-stained, near anus.

Bladder.—Healthy.

Lymphatic Syst.—Mesenteric glands size of small peas, also those in neck and other areas. No large glands on post. Abdom. wall enlargement slight therefore.

Neck.—Nothing abnormal.

Head.—Calvarium thin. Dura not adherent. Fluid not now in excess after the L. puncture of yesterday. Distinct flattening of convolutions on vertex. Vessels over vertex and other surfaces of brain full of dark blood congested, also those over cerebellum. Capillaries also slightly congested. Pia arachnoid clear in consistence (slight excess of fluid), colour slightly turbid. Strips easily without erosion. Lat. ventricles distended fluid in excess, very slight turbidity. No other macroscopic change in other parts of brain. Presented as a whole general features of Sleeping Sickness. Congestion of vessels, otherwise macroscopically, nothing.

Histological Examination.—Spleen and Liver, old malarial pigment.

Brain.—Presents a well marked meningeal exudation more marked than usual. Infiltration round vessels also well marked. *Pons* and *Medulla*, well marked infiltration round vessels in substance. *Cord.*—Many vessels show the usual exudation around. Meningeal surf., well marked infiltration. *Heart Muscle.*—Markedly degenerated in substance, also several areas of leucocytic infiltration especially round the vessels. Skeletal muscle gastroenemius, degenerated fibres not clear and separated from each other.

Case 15.—Alfonse. 15 M. Ad. Sept. 8, 1902. Still in hospital. Native of Kesube :—

Fam. Hist.—F. l. M. l. h. B. l. h. S. 0.

Pres. Hist.—At R. C. Mission, did work as a boy of light nature.

Prev. Ill.—Has had colds and some fever.

State on Ad.—Heavy drowsy look, heavy lips, trace of jaundice in conjunctiva. Very faint tremor in tongue, none in arms. Skin dry and harsh. No eruptions or pruritus. A large mass of glands in left post. triangular, smaller glands in other p. triangular and in ant. and sub-maxillary. Epitroch. not palp. axillary and groin slight. Speech slow. Walk impaired, slight shuffling gait.

Circ. System.—P. 100. Reg. equal, Vol. mod., tens, mod. V. W. unthickened.

Heart.—A. B. 4th sp. 3 inches from m. l. u. b. 3rd rib, l. b. 4 inches from m. l. R. C. $\frac{1}{4}$ inch to rg. of m. l. *Ausc.* Sounds clear and distinct, no murmurs.

Resp. System.—No cough. No sputum. R. 16. Reg. equal., cost. abdomen. Chest well formed. *Perc.* Reson. note all over. Ausc. breathing vesic., no prolongation of expiration.

Aliment. System.—Tongue, large, flabby, slight fur. Teeth good, tonsils not enlarged. Appetite good, bowels constipated. Abd. shows nothing on inspection. *Liver*, u. b. 5th and 6th l. b. c. m. not palp. *Spleen*, u. b. 7th, l. b. 12th at c. m. in mam. line not palp.

Urinary System. No albumin, blood, or sugar. No deposit.

Lymphatic System.—Many enlarged glands (one size of large almond on left side) in both p. triangles. A few slight in Ant. triangles. None sub-max., or sub-mental, or in groins. Epitroch and axillary not palp.

Integ. System.—Skin lost its gloss. No pruritus or eruptions, dry.

Nervous System.—Heavy drowsy look with a frightened aspect. Speech fairly quick, does not sleep much. No cranial nerves paralysis.

Motor Functions.—No emaciation or wasting. Muscular nutrition good. Faint tremor in tongue, grip good. No incoordination.

Sens. Functions.—No impairment.

Reflex Functions.—Superficial, all present, brisk. Knee not exaggerated, about ordinary, equal on both sides. Organic normal. No clonus. *Eye.* Pupils equal. mod. dilated, react to light and accommodation. No nystagmus. Hearing and smell unimpaired.

Further Notes.—September 23. Very little change. Tremor in tongue slight. Still heavy expression.

September 25. Seized with acute pain in abd., bowels constipated. Pressure over region of stomach causes patient to cry out with pain. Hot fomentations applied.

September 26. Pain in abdomen gone—better.

September 29. Drowsy, but does not show much change.

October 5. In *statu quo.*

October 10. About the same, brighter if anything.

October 20. About the same.

October 28. Appears a little better. No drowsiness. Goes about out of doors all day.

In conclusion, we have to tender our best thanks to Colonel Sadler, His Majesty's Commissioner of Uganda; to Dr. Moffat, C.M.G., Principal Medical Officer of Uganda; to Drs. Bagshawe, Hodges, Wiggins and Sly, for their kindness and help in forwarding our work in every possible way, and for much useful information and material.

Appendix.

FILARIA PERSTANS AND ITS RELATIONSHIP TO SLEEPING SICKNESS.

By GEORGE C. LOW, M.A., M.B., C.M.

Following the instructions issued by the Royal Society, namely, that Sleeping Sickness might be a disease due to the nematode parasite *Filaria perstans*, I took up this part of the work specially, and finally decided that there was no connection between the two.

This hypothesis was first advanced by Sir Patrick Manson, who demonstrated the presence of this parasite in three cases of this disease which were brought to England, one under the care of Sir Stephen Mackenzie at the London Hospital, and two at Charing Cross Hospital. Having just returned from the West Indies and British Guiana, where I had been making a special study of the different forms of *Filaria*, I had the advantage of possessing a vast amount of material bearing directly on this question before I started for Africa.

On arriving in Uganda, the method adopted to determine whether *Filaria perstans* could be incriminated in the production of Sleeping Sickness was to examine thousands of individuals' blood in different areas and to see if the parasite gave rise to any pathological changes. The special procedure was: (*a*) the examination of sick and sound in areas where Sleeping Sickness was rife; (*b*) the examination of people in areas where Sleeping Sickness had not appeared or had never been known; and (*c*) pathological data.

(A) *Areas with Sleeping Sickness.*

1. *Sese.*—This, a group of islands in the lake, was chosen on account of its convenience, and on account of the large amount of the disease present there. An examination of sixty-eight such cases showed that 66 or 97 per cent. had *Filaria perstans*, but against this, on the other hand, eighty-six individuals out of a hundred, carefully selected as free from Sleeping Sickness, also exhibited the same parasite in their blood.

2. *Entebbe and Neighbourhood.* An examination of fifty well-marked cases of Sleeping Sickness showed 45 or 90 per cent. with *Filaria*

perstans in their blood, whereas 100 ordinary individuals only showed *Filaria* in fifty-two, or 52 per cent.

In both these instances it is at once seen that there is a greater percentage of *Filaria* in the Sleeping Sickness cases than in the ordinary apparently healthy individuals. It is quite conceivable, however, that persons in the marasmic condition of this complaint may more easily become infected with parasites than those in health, and it must further be stated that in this latter estimation five of the Sleeping Sickness cases did not show *Filariæ* in their blood on the first examination, and then only one or two in 5 c.mm. of blood on further examinations. Of the 100 healthy individuals only one examination of 5 c.mm. of blood from each was possible.

3. *Kavirondo and the Kavirondo Islands.*—Dr. Hodges had already shown, before the commission arrived in Uganda, that, though Sleeping Sickness was very prevalent in the Kavirondo Islands, *Filaria perstans* on the other hand was very rare. In Nyala only three out of thirty-four cases of the disease showed the parasite, and in normal individuals only one out of fifty. Again, in Sigula Island, out of forty-five cases only three exhibited the parasite, and keeping up the contrast only two out of fifty-two healthy cases. Still more striking were the figures of Dr. Wiggins, of Kisumu, who found that not one out of 150 Wasemi cases of the disease had *Filaria perstans* in their blood, the same condition applying to the general healthy Kavirondo population of Kisumu, a place situated at the extreme east of the lake. A special visit to this place on my way home to England confirmed Dr. Wiggins' report as to there being no *Filariæ* there, yet a census, which Mr. Hobley, the Sub-Commissioner, kindly made for me, showed that there were seventy cases of Sleeping Sickness within 2 miles of his house at Kisumu (October, 1902). The disease at that time was spreading amongst the Kavirondo with great rapidity, and large numbers had died of it.

(B) *Areas Without Sleeping Sickness.*

1. *British Guiana.*—A few assertions, not supported by any evidence whatsoever, have been made that Sleeping Sickness has been seen in British Guiana amongst the Buck Indians. It has been said that the Indians when they fall sick take to their hammocks and lie there till they die. That they should do so is not by any means wonderful, as their hammocks are their beds, but this does not prove in the least that they are suffering from Sleeping Sickness. While travelling in the forests of the interior of British Guiana, I specially looked out for this disease, but though I often saw sick Indians lying in hammocks, I found that they were generally suffering from malaria and other diseases and never from Sleeping Sickness. Mr. Perkins, Acting Commissioner of Gold Mines, who has journeyed extensively through

the forests of the interior, informed me that the Indians were unaware of such a disease, and he personally had never heard of it. Again, though the Medical Staff in Georgetown, the capital of British Guiana, have been on the outlook for this malady for the last six years, no case has even been seen. The disease, therefore, I am convinced, does not exist in British Guiana. An examination of the blood of these aboriginal Indians, however, reveals the fact, already pointed out by Daniels and Ozzard, that *Filaria perstans* is exceedingly common, no fewer than 50 per cent. of them having it, some with it alone, while others very often have a sharp-tailed variety also, a different species. These nematodes seem to give rise to no symptoms.

2. *Unyoro.*—An examination of 100 individuals' blood from this large district near the Albert Lake, made by Dr. Moffat, C.M.G., and myself, gave seventy-four infected with *Filaria perstans*. As in British Guiana, Sleeping Sickness has never been known there.

3. *The Upper Nile Districts.*—Sleeping Sickness is unknown in all the Upper Nile stations. The percentage of filariasis is much lower here, for example 8·4 per cent. in the Alurs from Wadelai, but still it exists.

Conclusions.

The results of these examinations show :—

1. That there are areas where Sleeping Sickness exists and *Filaria perstans* is absent.

2. That there are areas where *Filaria perstans* is very common and yet there is no Sleeping Sickness.

3. That there are areas where both Sleeping Sickness and *Filaria perstans* are found, the parasite existing, however, in the blood of the healthy as well as in that of the sick.

Double infections of the blunt and sharp-tailed nematodes, *Filaria perstans* and *Filaria demarquaii*, are very commonly met with in British Guiana, but single infections of either may occur. Out of 163 aboriginal Indians examined at one place, there were thirty-eight with double infections, fifty-six with *Filaria perstans* alone and eleven with *Filaria demarquaii* alone. The average number affected with *Filaria perstans* is from fifty to sixty per cent. (C) pathological data which render it unlikely that *Filaria perstans* is the cause of Sleeping Sickness.

The occurrence of *Filaria perstans* in such large numbers of healthy people, both in areas with Sleeping Sickness and in those without, show that this worm probably produces no pathological symptoms whatever. It has been contended that because *Filaria nocturna* does not always cause pathological symptoms, that the same may be the case with *Filaria perstans*. One has only to look for a moment at the sites of selection in the human body of those two nematodes to see

Table showing the Prevalence of *Filaria perstans* in some Areas.

AFRICA.

No.	Race.	District.	No. of healthy individuals examined.	Percentage with *F. perstans*.	Percentage with *F. nocturna*.	Presence or absence of Sleeping Sickness.
1	Baganda	Entebbe	100	51	Not determined	Very prevalent.
2	„	„	100	42·2	2·2	„
3	„ (children under ten)	„	100	14·2	0	„
4	Baganda	Bulemezi	100	62	Not determined	A few cases.
5	„	Ndeji Bulemezi	100	27	„	A few suspicious cases (Mr. Leaky).
6	„	Kago	100	62	„	A few cases (Cook).
7	„	Mugema	100	50	„	?
8	„	Buddu	100	20	„	A few cases reported.
9	„	South Buddu	100	30	„	Absent (Bagshawe).
10	„	Gimbo Chagwe (examined by Moffat)	100	90	„	Very prevalent (Moffat).
11	Wasese	Sese	100	86	„	„
12	Bavuma	Buvuma Island (off coast of Usoga)	50	78	„	„
13	„	Bugaia Island	50	2	„	Prevalent.
14	Kavirondo	Kisumu	30	0	„	Very prevalent.
15	„	„ (examined by Dr. Wiggins)	100	0	„	„
16	Wasemi	Nr. Kisumi (examined by Dr. Wiggins)	150	0	„	„
17	?	Ankole (examined by Moffat and self)	140	7·8	„	Not known.
18	Wanyora	Unyoro „ „	100	74	„	„
19	Alur	Wadelai (examined by Bagshawe and self)	50	8·4	„	„
20	Nubians	Nile districts	100	6	2	„
21	Swahili	Coast	100	2	26	„
22	Europeans	Entebbe, &c.	20	0	Not determined	Very prevalent.

Table showing the Prevalence of *Filaria perstans* in some Areas—*contd.*

BRITISH GUIANA.

No.	Race.	District.	No. of individuals examined.	Percentage with *F. perstans* or *F. demarquaii*.	Percentage with *F. nocturna*.	Presence or absence of Sleeping Sickness.
23	Carib and Arawak Indians	Pomeroon River, British Guiana.....	100	71	Not determined	Absent.
24	Carib Indians	Demerara River.....	10	60	,,	,,
25	Warau Indians ...	Waini River.....	30	60	,,	,,
26	Akawoio Indians..	Barima River.....	20	36·6	,,	,,

that this does not follow at all. The adult or parental forms of *Filaria nocturna* live in the lymphatics, and the changes they give rise to are purely mechanical. Anything may at any time cause the death of the parasites, or a collection of a number may block the tube in which they lie, and consequent changes involving the lymphatic system are then apt to set up. In the case of *Filaria perstans* it is quite different; the adults here are living in the connective tissues of the mesentery, and all that could happen by their death would be perhaps a little local inflammation, though it is improbable that even this would take place.

The only other hypothesis would be (1) an accumulation of embryos in the fluids of the brain, which does not occur, or (2) the production of a toxin from the adult or embryo, an extremely improbable theory, as none of the other *Filariæ* are known to give rise to such products. *Filaria demarquaii*, which resembles *Filaria perstans* very closely in every respect, even to the site selected by the adults, gives rise to no pathological symptoms, and it is difficult to see by analogy why *Filaria perstans* should.

The final conclusions then one comes to, both from the distribution and the pathological anatomy, are that *Filaria perstans* has nothing to do with Sleeping Sickness, its presence in these cases being only a coincidence, as is the presence of *Ankylostoma duodenale*, *Ascaris lumbricoides*, *Trichocephalus dispar*, and *Bilharzia hæmatobia*.

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